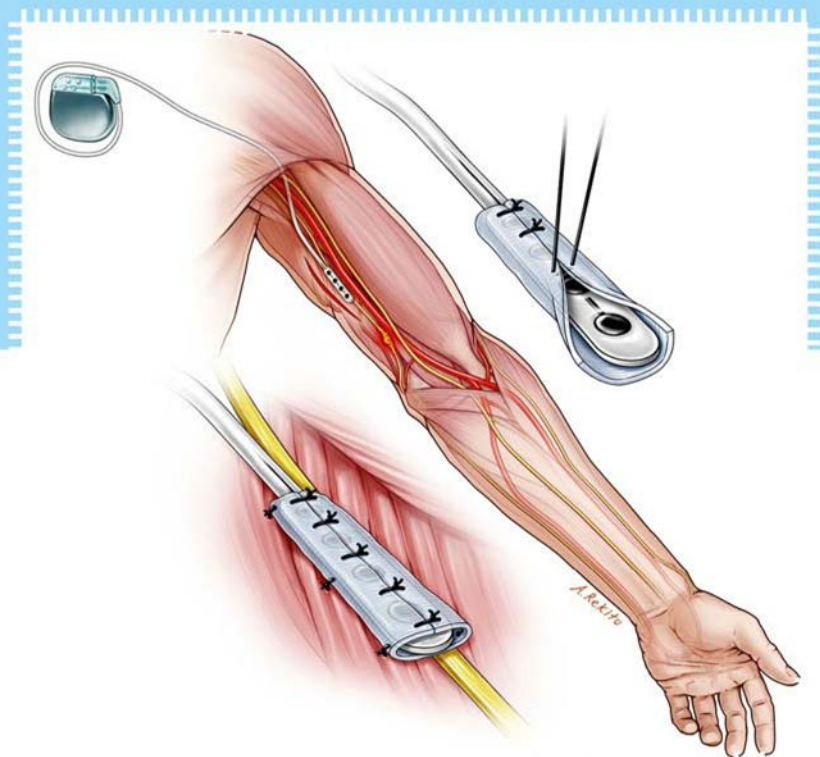


Handbook of Pain Surgery

Kim J. Burchiel

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Handbook of Pain Surgery

Kim J. Burchiel, MD, FACS

John Raaf Professor and Chairman Emeritus

Department of Neurological Surgery

Professor

Department of Anesthesiology and Perioperative Medicine

Oregon Health and Science University

Portland, Oregon

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Thieme Publishers Delhi
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Uttar Pradesh, India
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To Professor Yücel Kanpolat

Brother and Mentor

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Preface

When the textbook *Surgical Management of Pain* was first published in 2002, my goal was to provide a compendium of knowledge in the area of pain surgery that would both organize our approach to difficult problems, and to open a discussion on the strengths, weaknesses, and controversies that constitute this field. I believe that goal was achieved in the first edition. The second edition purposely omitted what I believed were outmoded procedures, and conceptual inconsistencies, but retained the original objective of focusing attention on the possibilities of surgical pain management.

The field of pain surgery will obviously continually evolve, but only with continued critique and honest rendition of outcomes can we hope to improve practice. It is within this framework that Thieme has graciously undertaken this current paperback edition—*Handbook of Pain Surgery*—the goal being to place this work into more hands, to inspire the next generation of practitioners, and to continue this important discussion. I am grateful to the publishers for this effort, and hope that the reader will question everything in this book. Truly, “better is possible.”

Kim J. Burchiel, MD, FACS
May 2017

Contributors

Feridun Acar, MD

Professor of Neurosurgery
Chairman, Department of
Neurosurgery
Functional Neurosurgery Unit
Pamukkale University
Vice-President
International Basic Neurosurgery
Course
Denizli, Turkey

Nicholas Barbaro, MD

Professor and Chairman of
Neurological Surgery
Indiana University School of Medicine
Medical Director, IUH Neuroscience
Center
Goodman Campbell Brain and Spine
Indiana Health Methodist Hospital
Indianapolis, Indiana

Giancarlo Barolat, MD

Director
Barolat Neuroscience
Presbyterian/St. Luke's Medical Center
Denver, Colorado

Allan J. Belzberg, MD, FRCSC

Associate Professor of Neurosurgery
Johns Hopkins School of Medicine
Baltimore, Maryland

Jeffrey A. Brown, MD, FACS

Neurological Surgery, PC
Great Neck, New York

Kim J. Burchiel, MD, FACS

John Raaf Professor and Chairman
Emeritus
Department of Neurological Surgery
Professor
Department of Anesthesiology and
Perioperative Medicine
Oregon Health and Science University
Portland, Oregon

Justin S. Cetas, MD, PhD

Chief of Neurosurgery
Portland VA Medical center
Assistant Professor
Department of Neurological Surgery
Oregon Health and Science University
Portland, Oregon

Grace Chen, MD

Assistant Professor
Department of Anesthesiology and
Perioperative Medicine
Oregon Health and Science University
Portland, Oregon

Cletus Cheyuo, MD, PhD

Research Assistant
Department of Neurosurgery
Buffalo General Hospital
Buffalo, New York

Daniel R. Cleary, MD, PhD

Department of Neurology
Yale School of Medicine
New Haven, Connecticut

Selçuk Göçmen, MD

Department of Neurosurgery
Pamukkale University
Denizli, Turkey

Viraat Harsh, MBBS

Department of Neurosurgery
Baylor College of Medicine
Houston, Texas

Jason D. Hill, MD

Indianapolis, Indiana

Yücel Kanpolat, MD

Emeritus Professor of Neurosurgery
Ankara University School of Medicine
Ankara, Turkey

**Douglas Kondziolka, MD, MSc,
FRCS(S), FACS**

Professor of Neurosurgery
New York University Langone Medical
Center
New York, New York

Elliot S. Krames, MD

Medical Director
Pacific Pain Treatment Center
Past President
International Neuromodulation
Society
Founding Editor
Neuromodulation, Journal of the INS
San Francisco, California

Shih-Shan Lang, MD

Department of Neurosurgery
University of Pennsylvania
Philadelphia, Pennsylvania

John Y. K. Lee, MD

Assistant Professor
Department of Neurosurgery
University of Pennsylvania
Philadelphia, Pennsylvania

Robert M. Levy, MD, PhD

Director
Marcus Neuroscience Institute
Boca Raton, Florida

L. Dade Lunsford, MD, FACS

Lars Leksell Professor of Neurological
Surgery
Distinguished Professor of
Neurological Surgery
University of Pittsburgh
Director, Center for Image Guided
Neurosurgery
Director, Neurosurgery Residency
Program
Chair, Technology and Innovative
Practice Committee
University of Pittsburgh Medical
Center–Presbyterian
Pittsburgh, Pennsylvania

Neal Luther, MD

Neurosurgeon
New Hampshire NeuroSpine Institute
Bedford, New Hampshire

Jonathan Miller, MD

Director, Functional and Restorative
Neurosurgery Center
Associate Professor of Neurological
Surgery
George R. and Constance P. Lincoln
Endowed Chair
University Hospitals Case Medical
Center
Case Western Reserve University
School of Medicine
Cleveland, Ohio

Charles Nelson Munyon, MD

Department of Neurological Surgery
Case Western Reserve University
Cleveland, Ohio

Haring J. W. Nauta, MD, PhD, FACS

Professor of Neurosurgery
Department of Neurological Surgery
University of Louisville
Louisville, Kentucky

Richard B. North, MD

President
The Neuromodulation Foundation, Inc.
Professor of Neurosurgery,
Anesthesiology, and Critical Care
Medicine (ret.)
Johns Hopkins University School of
Medicine
Baltimore, Maryland

Richard K. Osenbach, MD

Neurosurgeon
Department of Medicine
Pinnacle Health Neurosurgery and
Neurosciences Institute
Harrisburg, Pennsylvania

Elliot Palmer, MD

The Vancouver Clinic
Vancouver, Washington

Sunil J. Panchal, MD

President
National Institute of Pain
Tampa, Florida

Tammy Penhollow, DO

American Board of Anesthesia
Board Certified in Anesthesiology and
in Pain Medicine

Ahmed M. Raslan, MD

Assistant Professor
Department of Neurological Surgery
Oregon Health and Science University
Portland, Oregon

**Joshua M. Rosenow, MD, FAANS,
FACS**

Director of Functional Neurosurgery
Associate Professor of Neurosurgery,
Neurology, and Physical Medicine and
Rehabilitation
Northwestern University Feinberg
School of Medicine
Chicago, Illinois

Marc Sindou, MD, DSc

Professor of Neurosurgery
Department of Neurosurgery
Hopital Neurologique P. Wertheimer
University of Lyon
Lyon, France

Konstantin V. Slavin, MD

Professor
Department of Neurosurgery
University of Illinois at Chicago
Chicago, Illinois

Brett R. Stacey, MD

Medical Director
Comprehensive Pain Center
Professor
Department of Anesthesiology and
Perioperative Medicine
Division Chief, Pain Medicine
Oregon Health and Science University
Portland, Oregon

Nathaniel D. Stetson, DO

Neurosurgery
Neuroscience Specialists
Oklahoma City, Oklahoma

Daryoush Tavanaiepour, MD

Assistant Professor and Associate
Chair
Department of Neurosurgery
University of Florida College of
Medicine
Jacksonville, Florida

Ashwin Viswanathan, MD

Director of Functional Neurosurgery
Assistant Professor of Neurosurgery
Baylor College of Medicine
Houston, Texas

Phillip Weidner, DO

Medical Director
Comprehensive Pain Management
Center
Alaska Native Medical Center
Anchorage, Alaska

Richard L. Weiner, MD, FACS, FAANS

Vice Chair

Department of Neurosurgery

THR Presbyterian Hospital of Dallas

Clinical Associate Professor of
Neurosurgery

University of Texas Southwestern

Medical School

Dallas, Texas

William D. Willis, MD, PhD

Professor Emeritus

Department of Neuroscience and Cell
Biology

University of Texas Medical Branch

Galveston, Texas

Karin N. Westlund, PhD

Professor

Department of Physiology

University of Kentucky

Lexington, Kentucky

Section I

Pain Medicine

1

Approach to the Patient with Chronic Pain

Joshua M. Rosenow

Evaluating patients with chronic pain can be a challenging exercise for even the experienced clinician. These patients have often traveled a long road through the health care system and have experienced many frustrations. Moreover, they frequently present with expectations that may be difficult to meet. This chapter is intended to serve as an overview for neurosurgeons of some of the considerations that need to be taken into account when treating these patients. This process may be time-consuming, but it is time well spent because a hasty encounter will leave many things unresolved on both sides of the physician–patient relationship.

■ Prior to Arrival

A letter from the patient's referring physician may provide valuable insight into the patient's state of mind as well as his or her pain complaint. In addition, this helps to focus the encounter because these patients may have numerous somatic complaints. Moreover, it establishes a line of communication with the referring physician and aids in the coordination of care.

The physicians should obtain as complete a record of prior treatments and outcomes as possible, including operative reports, psychological reports, imaging, diagnostic testing (electromyography, discography, etc.) reports, imaging studies and reports (preprocedure and postprocedure for each surgery), specialist consultations, physical therapy summaries, and relevant office notes. It is also helpful to know if there have already been applications for long-term worker's compensation disability or social security disability, as well as if litigation is involved.

It may be helpful to send a set of clinic policies and procedures to patients prior to the visit to help establish ground rules on such items as missed appointments, medication prescribing and refills, general time frame for routine return phone calls, and paperwork completion.

■ The Initial Visit

It is crucial to understand the patient's goals not just for the initial encounter, but for the physician–patient relationship as well. Conversely, patients need to understand the capabilities and limits of the relationship (which parts of care the evaluating physician may or may not be willing to assume responsibility for) to

avoid misunderstandings down the road that can erode the patient's trust in the physician. For example, a patient may expect the evaluating physician to assume prescribing responsibility for chronic narcotics or to aid in the patient's quest for disability benefits.

Patients with chronic pain have already undergone numerous evaluations and explained their story ad nauseum, and may become disenchanted or angry at the thought of having to go through it again. They should be reassured that this is a key part of determining both why their prior treatments have not succeeded and if there are any further surgical treatments that may provide them with their desired outcome. Also, the patient should understand that it may require more than a single visit for a full evaluation and to determine a therapeutic course of action. This may also require further diagnostic testing or other consultative evaluations, such as psychological testing.

The failure of previous medical or surgical attempts to relieve pain may be due to the failure of the treatment itself, but it might also be related to a variety of underlying psychosocial issues. Pain relief that is judged as adequate by a previous physician may not have been helpful enough for the patient.

Possible reasons for prior treatment failure are:

- Incorrect diagnosis
- Incorrect choice of therapy (medical or surgical)
- Incorrect application of therapy (wrong level, wrong site)
- Failure to apply the therapy to the full extent necessary for symptomatic relief (e.g., declaring a medication to have failed prior to increasing the dose to a sufficient amount or not sufficiently decompressing a stenotic spine)
- Side effects limiting full application of a treatment
- Treatment-related side effect (adjacent segment degeneration, etc.)
- Patient factors (health, anatomy, allergies) that prevent full application of a treatment
- Intraoperative complication
- Technical failure of procedure (e.g., nonunion)
- Incorrect expectations of outcome (either on the part of the patient or the result of poor expectation management on the part of the physician)
- Medicolegal factors
- Occupational factors
- Psychological and social factors

■ History

General Aspects

A detailed history may be the most helpful factor in establishing a diagnosis and forming a treatment plan. The history provides important information not only about the possible mechanisms and pathophysiology of the patient's pain, but also about the emotional and psychological status of the patient. It often requires a combination of a firm hand to guide the interview and prevent excessive wandering by the patient, along with allowing enough flexibility to allow the patient

to volunteer valuable information. Keeping each of these sometimes complex patient histories chronologically organized is a good way to stay on track.

Pain History and Onset

Once the current baseline is established, including standard factors such as location, quality, radiation, duration, and exacerbating and alleviating factors, this pain may be analyzed in relation to the patient history.

Determining the relationship of the current pain to the original pain enables the physician to determine if this pain represents new pain or a continuation/progression of a previous pain complaint. What prior treatments were used? What effect on the pain did they have? This helps determine if prior therapies were inappropriate (perhaps guided by wrong diagnosis) or just not used to their full extent.

If the current pain is substantially different from the original pain, this may imply that a different process may be responsible for the current pain or that the current pain is the result of a treatment side effect. Patients often undergo misguided treatments for pain syndromes. These treatments can themselves cause further pain. As previously stated, procedural complications can also result in pain that is unlike the presenting pain. The physician should be cautious of those patients whose pain changes significantly in location and character after a treatment.

Was there an obvious inciting incident associated with the pain? Was this mechanism consistent with the patient's original complaints? The location, distribution, quality, intensity or severity, and duration of the first pain should be ascertained. Were there any other, associated neurologic symptoms at the time and did any of these develop in a subacute fashion? Was any treatment applied immediately?

Pain that is the result of an activity on the job presents a special situation due to the issue of worker's compensation claims. This type of history needs to be even more fastidiously documented, including the exact time and date of the injury. Other information should be obtained, such as whether the patient continued to work after the injury, when and to whom the injury was reported, and the response of the patient's employer to the injury as regards the patient's work status.

Neuropathic pain syndromes, such as complex regional pain syndrome (CRPS 1 and 2), most often have an antecedent injury that sets the syndrome in motion (with trigeminal neuralgia—Burchiel TN 1—being an exception), unlike nociceptive pain, such as degenerative spinal disease, which is often slowly progressive.

The physician should also ascertain the impact of the pain on the patient's daily life. This is accomplished by asking the patient to provide the timeline of an average day. This provides important insight into the psychological effects of the pain on the patient's life and the level of disability the patient is experiencing.

Pain Characteristics

The patient should be asked to describe in detail the current quality, site, radiation, severity, and alleviating/exacerbating characteristics of the pain at the time of evaluation and to indicate whether any of these factors has changed since the onset or during the interval. Those factors that have no effect on the pain are important as well. Such factors include stress and other emotional disturbances, movement,

pressure, heat or cold, coughing, sneezing, straining, and deep breathing. The 0 to 10 visual analogue scale (VAS) is the most commonly used pain rating scale, even if it is not the most robust measure. It is often more useful to administer the visual analogue scale by asking the patient to mark the level of pain on an unlabeled 10-cm line. Physicians should be wary of those patients whose pain either never varies from 10 or is rated as “11” on a scale of 0 to 10.

Pain may be classified as localized, radiating, or referred. Localized pain continues at the same location as its origination. This pain may be associated with other anatomic and sensory changes at the site of pain, such as hyperalgesia, wind-up hyperpathia, color change, and trophic changes of the skin. Pain may also be described as radiating along the distribution of a nerve root's dermatome or the innervation of a peripheral nerve. This is distinct from pain that is referred from a deep visceral structure to a separate distinct anatomic location. Classic examples of this are back pain or superficial abdominal pain from chronic pancreatitis and scapular pain from cholecystitis.

Pain Treatment History

Obtaining a recounting of past medical and interventional treatments and the response to each is an invaluable part of the evaluation. A thorough analysis of this part of the history prevents the clinician from repeating past failures. Which classes of medications have been tried? Were the medication trials adequate? Were interventional techniques used appropriately? Did each subsequent treatment follow logically based on past information? Has *every* treatment failed to provide any relief? Moreover, this history helps to ascertain patient compliance and assess for etiologies of past treatment failures. The clinician needs to take a very critical view of this section of the history, always asking *why* a treatment failed and if there is anything else for the neurosurgeon to offer the patient.

Past Medical and Surgical History

Concomitant medical conditions can adversely impact both medical and surgical treatments. Factors such as obesity, diabetes, hypertension, hypothyroidism, chronic obstructive pulmonary disease, and inflammatory arthritis are common and each has the ability to complicate therapy. Patients frequently will omit conditions they do not consider important or will omit conditions they consider treated. For completeness, the inquiry should proceed through a comprehensive list of organ systems.

If the patient has failed multiple surgical procedures for the pain syndrome, were there surgical failures for other conditions as well? If so, is this due to an intrinsic patient medical factor, poor surgical techniques, unrealistic expectations, the patient seeking unnecessary surgical treatments, or just bad fortune? Does the patient appear compliant with the physician instructions for other ailments?

Family History

Patterns may be recognized from a family history. Have multiple family members undergone spinal surgery? Are they all employed in high-impact professions? Is there a family tendency toward spinal degeneration or stenosis? Are multiple family members receiving disability insurance? Chronic pain behavior may be a

learned trait within families.¹ There is level III evidence through cohort studies that a history of child abuse may increase pain and pain behaviors.¹⁻⁴

Social and Psychological History

A complete psychological and psychosocial assessment is crucial for the overall management of the patient with chronic pain. There is substantial class III and IV literature on this subject that is well covered in other sources.⁵⁻⁹ The psychological and psychosocial part of the history helps to determine the contribution of affective or environmental factors to the patient's pain syndrome. Referral to a psychologist with special expertise in the evaluation and treatment of patients with chronic pain is an essential part of the total care of these patients. At the minimum this part of the history should include a listing of current and prior psychological and psychiatric illnesses. Special attention should be paid to depression, a common condition in patients with chronic pain. The astute neurosurgeon will delve deeper into this issue than simply inquiring about the patient's mood. The patient's daily schedule provides important data about his or her depression. Other clues are irritability, insomnia, abulia, weight gain or loss, and suicidal ideation. The patient's family can provide important perspective as well.

The neurosurgeon should also explore the patient's vocational status and vocational stressors, including compensation and litigation issues. These can impact the patient's motivation and outcome from treatment as demonstrated by level III and IV evidence.¹⁰⁻¹³

Whereas the history should include current prescription drugs, an appropriately thorough history should also include over-the-counter and alternative (e.g., herbal) medicines. Illicit pharmaceutical use (both current and past) should be elicited by direct questioning, including specifically asking about the most commonly abused pharmaceuticals. It is important to determine not only what patients are using, but also if they are using it appropriately. Be alert for patients using others' prescription medications for their own benefit or using medications up earlier than prescribed and obtaining substitutes illicitly. A history of tobacco smoking and alcohol use, including the type and frequency of use of each, should be included as well. Physical and psychological dependence on drugs and alcohol is a common impediment to treatment.

A useful exercise is to conceptualize the patient's pain syndrome as separate components of pain and suffering. Pain is the purely physical and physiologic component of the syndrome, whereas suffering includes the individual's reaction to the pain as well the pain's effect on the rest of the person's psychological and emotional environment. Suffering includes depression, anxiety, loss of self-esteem, failed relationships, past emotional and physical abuses that shape the pain response, poor coping strategies, and withdrawal from friends and family. Surgical interventions may be an excellent method for dealing with the pain, but they are inadequate at handling an impressive suffering component. Again, a skilled pain psychologist is adept at determining the balance between the pain and suffering components along with identifying maladaptive coping strategies and other pitfalls of which the surgeon should be aware prior to embarking on a therapeutic relationship with the patient.

Although it is true that there is a small population of individuals entirely without a physical basis for their pain complaints,¹⁴ the majority of patients with chronic

pain show a complex interplay between psychological and physical components of their pain syndrome in varying proportions. Among the neurosurgeon's challenges in evaluating and managing patients with chronic pain are first determining the relative balance between these factors and then using that estimation to drive the selection of an individualized combination of medical, interventional, surgical, and psychological treatments for each patient.

Physical and Neurologic Examinations

The examination of the patient with chronic pain is essentially no different from that of a patient presenting for any other type of neurosurgical evaluation. The neurosurgeon must be vigilant for signs such as weakness and pathologic reflexes that may indicate conditions such as nerve root or spinal cord compression that could warrant urgent further evaluation or treatment. These findings should not be discounted or neglected just because the patient may have already seen other physicians or have had other surgical procedures.

The central sensitization that occurs in many patients with chronic pain can result in certain characteristic exam findings. Central sensitization affects the wide-dynamic-range (WDR) neurons in the dorsal horn of the spinal cord that receive input from both Ab and C fibers. In neuropathic pain states, Ab fiber depolarization also results in stimulation of these other fibers, resulting in allodynia, the perception of pain from light touch. Patients with chronic pain may exhibit generalized hyperpathia, exaggerated and prolonged reactions to painful stimuli. For example, this may manifest itself as the patient reporting that a pin prick is intensely painful over the entire body. Repetitive stimulation of C fibers may result in an augmented response to each subsequent stimulus, a process known as wind-up. Repetitive light stroking of the painful area is interpreted by the patient as increasingly painful with each iteration.

Red flags on examination should engender skepticism in the examiner. These may include Waddell's signs^{15–17} for nonphysiologic back pain (mostly level IV evidence) and other inconsistencies on physical and neurologic examination. For instance, does a patient with apparent ankle dorsiflexor weakness when seated have the ability to heel-walk without much difficulty? Do the findings fail to conform to a peripheral or spinal nerve distribution? Importantly, Fishbain¹⁸ published a structured review (level III evidence) of the medical literature on the validity of Waddell's signs and noted that Waddell's signs are not reliable as a discriminator of physiologic from nonphysiologic pain.

■ Formulating a Treatment Plan

The patient with chronic pain requires an individualized treatment plan. First, the treatment team should be able to work in a cohesive manner and present a united front to the patient. Representative members of a pain team are chosen from the following clinical areas:

- Surgical specialist(s)
- Anesthesia
- Neurology

- Medical specialists (internal medicine, oncology, etc.)
- Psychiatry
- Pain psychology
- Psychiatry
- Nursing
- Physical therapy
- Occupational/vocational therapy
- Social work
- Addiction medicine

The clinicians involved should have clear and open lines of communication between them. The best method for achieving this is to set up a regularly scheduled patient management meeting attended by the pain team. A team consensus may be reached and be discussed with the patient and his or her family.

Next, in defining a plan, it is important to outline the plan in as much specificity as possible and then stick to it. The plan should have steps included for dealing with medication-related side effects and procedural failures. This gives the patient a clear understanding of what steps will be taken and in what order. Moreover, the patient will understand what the expected outcomes are for each step and what will be done if the actual result does not meet the expected outcome at each step. Part of outlining this plan may include the signing of a treatment contract on the part of the patient and clinician.^{19–21} If a contract is signed, the patient should have a copy to keep. Contracts should denote the obligations of both parties as well as the consequences for violations.

Setting expectations of treatment is a crucial part of the overall plan. Often the patient's expectations have not been met by past medical and surgical treatments either because the treatments have not delivered anticipated results or because the patient's expectations were not realistic. Substantial time should be spent discussing realistic outcomes for each care step, as well as for the overall plan of care. For instance, it may or may not be realistic for a patient to return to the same line of work following treatment, but it is critical to understand if the patient believes that he or she will do so. The clinician's and patient's ideas of a "good" outcome may be divergent, and this disparity needs to be understood and managed.

Goal and expectation setting should include lifestyle modification as needed. Smoking and obesity are negative determinants of outcome^{22–24} (level IV evidence), and the willingness to work on resolving these problems is a good determinant of the patient's level of motivation. Return visits may need to be scheduled to evaluate progress on certain goals or to discuss consultation or imaging results prior to the physician's agreeing to embark on a therapeutic relationship. Return visits also allow the physician to assess the amount of information internalized by the patient from previous visits. Written materials given to the patient to read at home may aid in increasing retention of information discussed during the office visit.

Some patients may not be accepted for care. A true pain team is not just a dumping ground for all patients that other specialists do not want to manage. Patients with a significant ongoing pattern of pharmaceutical misuse, whether legal or illegal, need to first have these issues resolved. Moreover, severe overwhelming emotional and psychological problems also need to be brought under control, especially prior to nonurgent surgical procedures.

Editor's Comments

It is difficult to communicate how a clinician should approach a chronic pain patient. Experience clearly matters. What Dr. Rosenow has outlined here is an excellent summary that hits most of the important points. From my standpoint, there are a couple of other aspects of the assessment of a patient with chronic pain that are worth mentioning.

First, although a comprehensive review of the patient's history is vital, many patients with chronic pain appear for evaluation with literally reams of data: prior evaluations, treatments, diagnostic testing, imaging, and so on. The clinician must use the record and not be a victim of it. Only experience can teach which aspects of the record merit initial close scrutiny, and the record should be available indefinitely for re-review. However, in some cases a page-by-page reading of the records is simply not practical. It is important that the evaluating clinician take a fresh look at the problem, working from the present backward, and use the record to answer directed questions.

Next, remember that the patient has come to you for assistance. At the initial encounter, patients often bring anger, expectations, and demands, which may or may not be appropriate for the first minutes of your acquaintance. It is imperative for the patient to know you are there to try to help and to render an opinion, and perhaps therapy. You are not there as a fiduciary for whatever trauma the patient may or may not have sustained from dealing with the health care system, worker's compensation, or the legal process. I often have to remind patients that we have just met, and that I am there solely to try to alleviate their pain problems. This message can be conveyed, if necessary, in a forthright and nonthreatening manner. I find that this helps to establish the "ground rules," and in most cases, further cement the patient-physician bond.

One of the most important statements to make during the initial encounter with a patient with chronic pain is that you *believe* him or her. None of us has the capability to feel what the patient is experiencing. The patient may appear to have more pain than the diagnosis can explain, but we are only observers of the outermost shell of the nested layers of suffering, pain, and nociception in a given patient. Our job is to determine if we can help the patient by surgical or nonsurgical means, *not* to act in judgment over the patient's behavior. It is vital that the patient know this, and this statement, more than almost any other comment you can make to the patient, calms what may be a stressful situation, and establishes the roles of patient and caregiver.

When a patient comes to a surgeon, or at least, an interventionalist, the underlying question will be: "Would a surgical procedure help?" The surgeon should quickly assess whether either a neuromodulatory or destructive procedure would be indicated. In most cases, nondestructive neuromodulation is best. However, destructive procedures for trigeminal neuralgia or brachial plexus avulsion pain are very effective. On the other hand, a negative answer to the question of surgery should not be equated with failure. Many times eliminating that option focuses therapeutic strategy and energy on nonsurgical therapies. It is an important negative, and it may deflect the patient from yet another attempt at pain relief from what is a passive therapy.

The main challenge to the treatment of a patient with chronic pain is the willingness of the caregiver to engage. It is not an easy venture, as Dr. Rosenow points out, but it is one that can be fulfilling for the patient and clinician. Reading this chapter will set the clinician up to engage successfully in the care of these patients, an experience that will be seasoned over time.

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2

Management of Pain by Anesthetic Techniques

Elliot Palmer, Phillip Weidner, Grace Chen, and Brett R. Stacey

The specialty of pain medicine encompasses multidisciplinary care. This includes anesthetic techniques to diagnose and ameliorate pain, specialized rehabilitation to expand functional capacity, and psychological counseling to help patients cope with pain and improve overall quality of life, in spite of pain. This chapter focuses on procedural management techniques including axial and peripheral nerve blocks and other percutaneous techniques. Such procedures have many different purposes: diagnosis of the source of the pain, planning surgical intervention, facilitation of rehabilitation, treatment of acute conditions, and treatment of cancer-related pain. There are many retrospective cohort studies and anecdotal recounts of effective injections that support the long-standing tradition of injection procedures. However, the number of randomized-placebo controlled studies has been limited. In this chapter, we review commonly performed injection techniques and the literature supporting them.

■ General Standards for Periprocedural Care

As with any invasive procedure, performance of anesthetic interventions in chronic pain management includes nominal risks of bleeding, infection, and allergic reaction. Each procedure also carries its own potential complications. There are recent case reports of fungal infections associated with contaminated methylprednisolone epidural injections as well as more dated reports of spinal anesthesia following facet joint injection.^{1,2} Patients should be informed of these risks and potential complications, and consent should be obtained.

Bleeding diathesis, systemic infection, and localized infection in the region of the procedure are considered absolute contraindications because of the potentially devastating consequences of bleeding or infection proximal to the spinal cord. A history of allergic reaction to radiographic contrast is only a relative contraindication because it usually can be preempted by pretreatment with corticosteroids and H1 and H2 antagonists. Although patients occasionally report an allergy to a local anesthetic, true allergic reactions to local anesthetics are rare to the point of vanishing.

Basic precautions should be taken to reduce risks. If sedation is required, or the possibility for significant physiologic change is present, intravenous access should be obtained. For most procedures, it is appropriate for patients to take nothing by mouth before the procedure and to avoid driving home afterward. Fluoroscopy and ultrasound can be used to ensure accurate positioning of needles, as is discussed later in this chapter. Finally, the International Spinal Injection Society (ISIS) has published standards for performance of some procedures involving

spinal injection.³ At our institution we follow similar guidelines, which include physiologic monitoring of the patient and observation of aseptic technique with sterile skin preparation, drapes, and gloves. Sterile gowns are worn for intrathecal line placement; implantable pumps and spinal cord stimulators are placed in the operating room.

In addition to these general safety principles, guidelines should be observed in using such interventions appropriately. Before initiating an invasive diagnostic or therapeutic regimen, the natural history of a patient's complaint must be considered. For example, acute-onset back pain without associated neurologic findings typically resolves within 8 weeks, independent of any interventions.⁴ Because the discussed procedures do entail risks and can be difficult to interpret, ISIS recommends that any invasive diagnostic or potentially therapeutic interventions be avoided for at least 4 weeks from the onset of symptoms and 3 weeks after initiation of more conservative, noninvasive measures. A similar strategy is followed at our institution, although exceptions are made if the indications are strong and the severity of symptoms is likely to lead to prolonged inactivity and deactivation.

■ Imaging

Intraprocedural fluoroscopy, and more recently ultrasound, have become essential adjuncts for performing neural blockade techniques. Fluoroscopy and radiopaque contrast material help to improve the safety and accuracy of needle placement before regional anesthetic or neurolytic interventions are performed and to verify accurate neuroaxial catheter or spinal cord stimulator electrode placement. Ultrasound helps to visualize soft tissue densities, such as nerve plexus and tissue planes demarcated by fascia that fluoroscopy does not reveal.

The celiac plexus neurolytic block demonstrates the advantages of fluoroscopy compared with the blind needle technique, but also highlights a major deficiency of this approach. This block is commonly performed using a two-needle technique at the L1 level. The aorta lies anterior and slightly to the left of the anterior margin of the L1 vertebral body. The inferior vena cava lies to the right of midline, and the kidneys are posterolateral to the great vessels. The pancreas is anterior to the celiac plexus. The celiac plexus is anterior to the diaphragmatic crura and extends anterior to and around the aorta. With such crucial organs and vessels surrounding the celiac plexus, accurate placement of the two needles is essential. The left-sided needle is advanced until it lies just posterior to the aorta on the left, and the right-sided needle is advanced to the anterolateral aspect of the aorta on the right. Using fluoroscopy, a small volume of contrast material is injected through each needle, and its spread can be observed.

The use of fluoroscopy and contrast material should not be limited to anatomically complicated procedures only. Epidural steroid injections (ESIs) traditionally have been performed using a "blind" technique without fluoroscopic guidance. The blind technique introduces the potential for erroneous needle placement and subsequent injection of steroids into unintended areas, such as the intrathecal space, leading to possible adhesive arachnoiditis. White and coworkers^{5,6} found that inaccurate needle placement occurred in only 25 to 30% of blind injections, even in the hands of skilled and experienced proceduralists. Injecting variable amounts of radiologic contrast material under fluoroscopic observation before therapeutic ESI

potentially improves safety and efficacy. The risk of unintended intrathecal injection and its consequences can be virtually eliminated. Moreover, the traditional practice with the blind ESI technique to proceed with a second and third steroid injection as a routine series to assess efficacy becomes unnecessary.⁷ Documenting the distribution of injected materials also may explain a patient's response if a unilateral or limited epidural block is encountered. Finally, even with negative needle aspiration, a significant number of injections following blind needle placement have been shown to be intravascular.⁸ Intravascular needle placement is ascertained quickly by rapid uptake and disappearance of contrast material injected under fluoroscopy in continuous pulse mode. Needle placement can be corrected easily if this intravascular contrast pattern is visualized.

■ Peripheral Nerve Blocks

Local anesthetics interrupt nerve conduction by sodium channel blockade, thus decreasing transmission of sensory and motor information. Coupled with knowledge of innervation patterns and nervous system anatomy, techniques to deliver local anesthetics can be powerful tools for perioperative care for extremity and abdominal surgeries. To a lesser extent, this had been also used to treat chronic pain conditions or to facilitate rehabilitation. Transient pain relief persisting until the local anesthetic effect dissipates provides the basis for diagnostic blockades. The well-documented, frequently encountered, and not well understood phenomenon of prolonged pain relief after injection of local anesthetic is the basis for therapeutic injections. Prolonged pain relief may result from altered function in the affected area, altered central nervous system function, or some undescribed mechanism. Limited controlled studies, wide variations in technique, lack of standards, and inconsistent outcome assessment limit their acceptance in the era of "evidence-based medicine."

Peripheral nerves, such as the ilioinguinal nerve,⁹ intercostals nerve, and lateral femoral cutaneous nerve,¹⁰ that can cause chronic conditions have been injected with short-term good results. Neurosurgery referral for neurectomy or neuromodulation is appropriate after the efficacy subsides.

■ Epidural Steroid Injection

One of the most common types of injections performed by interventional pain specialists are epidural steroid injections. The procedure has been used to diagnose radicular and referred pain from specific levels of the spine and to treat disk herniation radicular pain related to neuroforaminal stenosis, spinal stenosis, and postlaminectomy syndrome.¹¹ Injections typically consist of both local anesthetic and steroid, although they are occasionally done with only one or the other.

Principles

Inflammation has been proposed as playing a key role in symptomatic nerve root irritation associated with herniated intervertebral disks.¹² Extruded nucleus pulposus material contains proinflammatory substances and produces an inflam-

matory response in the epidural space and in the underlying nerve roots. Pain and other symptoms are likely produced by a combination of this inflammatory response, edema, and the mechanical pressure on nerve roots.¹³ Additionally, degenerated, symptomatic areas of the spine demonstrate sensory nerve sprouting into the outer layer of abnormal intervertebral disks, vertebral endplates, and other structures, providing additional sources of spinal pain.¹⁴

Proinflammatory factors such as phospholipase A2 (PLA2) is released when the nucleus annulus is interrupted. This and other proinflammatory factors such as neuropeptide Y and VIP foster the inflammatory cascade and sensitize nerve endings that produce focal pain sensations in the disk and the surrounding nerve root.¹⁵ The goal of epidural injection is to deliver anti-inflammatory and analgesic medication to the spinal level most affected. Most commonly, this source is the disk and nerve root at the symptomatic level. The classes of medications injected—local anesthetic and steroid—are generally agreed upon, yet there remains a fairly wide variety of solutions, volumes, and delivery techniques used in modern clinical practice.¹⁶

It is well known that local anesthetics interrupt nerve transmission and thus can be effective for the transient relief of pain. Additionally, perineural inflammation accompanies many painful conditions, providing a rationale for including corticosteroids in these therapeutic injections. The mechanism of glucocorticoid activity is not yet fully understood. One mechanism of action is the altering of endothelial adhesiveness toward resting polymorphonuclear leukocytes. Glucocorticoids inhibit the display of chemotactic molecules on the surface of the endothelial cells, preventing leukocyte aggregation and minimizing endothelial injury, which is usually caused by cellular transmigration.¹⁷ Steroids have numerous anti-inflammatory and membrane-stabilizing properties that have been shown to decrease edema and sensitization as well as to provide pain relief when applied to the vicinity of painful nerves.

Epidural steroid injections are indicated primarily for the treatment of radicular extremity pain that has not responded to more conservative treatments. The goals of treatment include pain relief and ultimately improvement of functional capacity. The length of epidural steroid injection efficacy is variously reported, ranging from weeks to more than a year.¹⁸ Even short-term relief may provide the additional benefit of allowing patients to participate in physical therapy that would otherwise be prohibited by pain.

Epidural steroid injections are also commonly performed for pain related to lumbar central stenosis. The evidence for injections alleviating pain related to foraminal stenosis is better than that for central stenosis.¹⁹ Nevertheless, patients who are not appropriate surgical candidates may find this form of injection helpful, if not curative of the stenosis.²⁰ There is a paucity of randomized and controlled studies regarding the use of ESI to treat mechanical or muscular axial back pain.

The two basic approaches in widespread use are interlaminar and transforaminal injection techniques. The interlaminar blind approach to the epidural space is the one that most clinicians are familiar with and is frequently employed for both perioperative and labor analgesia. The landmarks are the spinous processes, which are often easily palpable and occasionally visible (**Fig. 2.1**). The only major differences in this technique when used for chronic pain states are the more widespread use of fluoroscopic guidance and the composition of the injectate. The interlaminar approach has the advantage of simplicity, delivering most of the in-

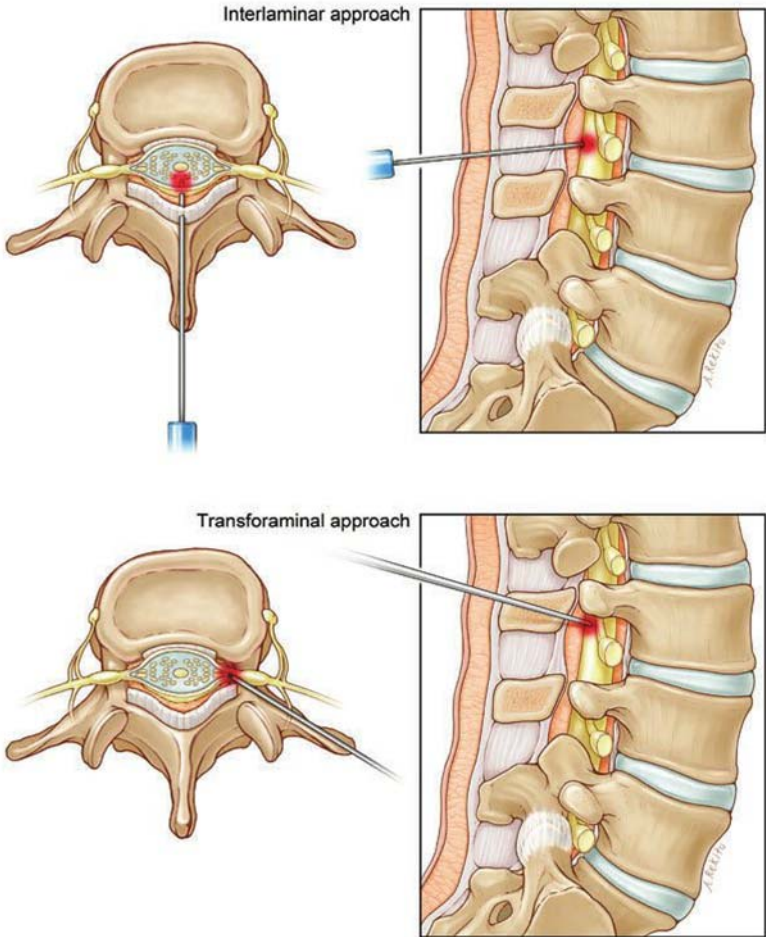


Fig. 2.1 Interlaminar epidural steroid injection. The needle is introduced percutaneously and traverses the supraspinatous ligament, the interspinatous ligament, and the ligamentum flavum.

jectate into the epidural space, but it has the disadvantage of delivering the medications into the center of the posterior epidural space rather than focusing the medication directly at the point of presumed pathology. The transforaminal approach, on the other hand, accesses the anterior epidural space at the level of the spinal nerve. Correct application of this technique requires the use of fluoroscopy because surface landmarks and tactile sensations are unreliable in ensuring appropriate final needle position. The primary landmarks for performing this injection in the lumbar area are the transverse process above the desired nerve root (best

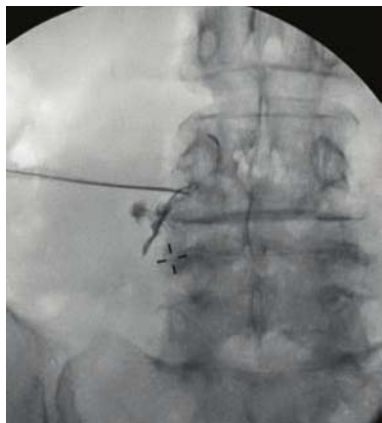


Fig. 2.2 This image shows a needle placed in the superior aspect of the right L5 neural foramen. Contrast dye is visible medial to the pedicle, indicating successful epidural spread.

viewed on anteroposterior [AP] projection) (**Fig. 2.2**) and the superior aspect of the nerve root foramen (best seen on lateral projection). We are aware of two additional approaches to neural injection, catheter-driven lysis of adhesions and spinal endoscopy, although neither is in widespread use and both are beyond the scope of this discussion.

Outcomes

The question of efficacy is an extremely complicated and multifaceted one. There exist a staggering number of studies of varying design and quality examining a wide variety of techniques, time courses, and end points. The resulting pool of outcome data is not cohesive and spans a range from a lack of statistically significant short- or long-term pain relief or functional capacity, to impressive improvement in

both—and including just about everything in between. For this reason, a complete and exhaustive review here is not practical. Instead, we have focused whenever possible on high-quality studies published in the decade prior to the release of this edition.

The best evidence behind ESI is in the treatment of radicular pain caused by a herniated intervertebral disk. Positive data from two relatively recent, prospective, randomized, placebo-controlled studies support the practice.^{21,22} Much attention has also been given to the question of possible differences in the efficacy of the two aforementioned injection techniques. Whereas superiority of the transforaminal approach has long been suspected and has been suggested by retrospective studies,²³ this assertion was not confirmed by another high-quality prospective, randomized study.²⁴ Although that study did not demonstrate superiority outright, it did suggest that results were similar despite a significantly lower dose of medications when the transforaminal route is employed.

The literature supporting ESIs for the treatment of painful spinal stenosis is sparser and consists mainly of retrospective case control studies and reviews. The best of these do suggest at least short-term improvement for both interlaminar and transforaminal approaches.^{25,26} Although no data are available at the time of this writing, a large-scale prospective randomized trial has been launched: the Lumbar Epidural Steroid Injection for Spinal Stenosis (LESS) study.

Facet Arthropathy and Radiofrequency Medial Branch Denervation

The lumbar and (later) the cervical, thoracic, and facet (or zygapophyseal) joints have been topics of considerable interest because they pertain to axial spinal pain and its minimally invasive treatment. In particular, the technique of reducing pain

caused by facet joint arthropathy by selectively denervating the facet joints has gained much support because of an increasingly positive body of evidence in the medical literature.

The human spine has facet joints at each level between the C1–2 and L5–S1 joints, inclusive. These are actual synovial joints, and the capsule and synovium of each are extensively innervated with sensory fibers. These include mechanoreceptors²⁷ as well as fibers containing substance P²⁸ and numerous other neurotransmitters linked to nociception.²⁹ The innervation generally is accepted to be segmental and based primarily on the medial branch of the primary posterior ramus of each segmental spinal nerve; however, more recent research has indicated that there is also nonsegmental and autonomic innervation of the facet joint.³⁰ The lumbar facet joint has been recognized³¹ as an important generator of axial spinal pain.

In addition to the facet joint capsule and its contents, the medial branch also innervates the multifidus muscle segmentally. This muscle is a significant source of pain in and of itself, and may account for some of the analgesia associated with lumbar medial branch blocks and denervations.³² A multifidus electromyogram also may be used as an outcome determinant in studies of lumbar medial branch denervation resulting from the specific innervation.

Cervical, Thoracic, and Lumbar Medial Denervation

In the late 1970s, Nikolai Bogduk and colleagues clarified the neuroanatomy of the facet joint. This, in combination with improvements in the technology available for radiofrequency neurodestructive procedures, led to increasing interest in the use of radiofrequency (RF) energy to produce lesions in the nervous supply of the facet joints. Although the definitive studies are in progress or in press at the time of this writing, initial work in the field indicates that this technique of treating axial lumbar pain is more frequently successful and less complicated than alternative means of treating pain mediated by lumbar facet arthropathy.

Radiofrequency Denervation

Various technical considerations led to the use of RF energy as the method of choice in the denervation of the medial branch (**Table 2.1**). Recent improvements in equipment include small-diameter (22 gauge) and curved probes to minimize tissue trauma and improve navigation (**Fig. 2.3**). The lesion generator, also used for intracranial functional neurosurgery, allows for multiple settings, depending on the procedure.

Outcomes

Numerous studies have demonstrated prolonged effects of RF medial branch denervation. The procedure in the cervical region is the most thoroughly studied. In this area, 75% of the treatment group had at least 50% analgesia for a median duration of 263 days.³³ A follow-up study of initial responders demonstrates a median analgesic duration of 422 days. In RF denervation, the neuronal contents are selectively coagulated, which interrupts neuronal function; however, the neuronal substrate remains.³⁴ This prevents neuroma formation but allows for regrowth of the nerve.

Table 2.1 Lumbar medial branch denervation comparison

Characteristic	Precision of lesion size	Collateral damage due to denervation	Trauma of procedure	Ability to assess intravascular status	Ability to stimulate adjacent nerves
Type of neurotomy					
Radiofrequency	5	5	5	5	5
Cryotherapy	5	3	2	0	5
Surgical	0–5	1–3	3	5	0–5
Injection of lytic chemicals	2	1–3	5	5	0

Note: The rating scale is from 0 to 5: 0 = undesirable; 1 2 3 4 5 = desirable.

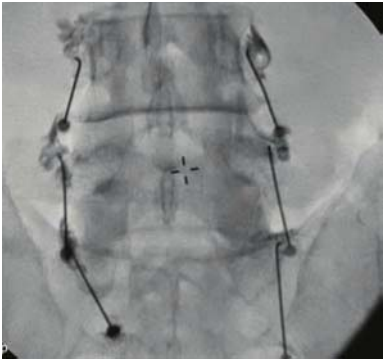


Fig. 2.3 Lumbar medial branch block under fluoroscopic guidance.

Thus far, the literature on this subject has studied medial branch denervation as monotherapy. There may be advantages to combining appropriate denervations with other multidisciplinary therapies, however, such as psychological and physical.

The Sympathetic Nervous System

The autonomic nervous system is composed of the sympathetic and parasympathetic nerve fibers, which work in concert to regulate the body's homeostatic functions. The sympathetic system is generally attributed

with vasoconstriction, increasing heart rate, decreasing intestinal motility, and piloerection, but it also maintains a significant role in the perception of pain and visceral sensations through C fibers. These are unmyelinated nerves whose cell bodies are in the dorsal root ganglia and enter the spinal cord via the dorsal horn. Sympathetically maintained pain is typically characterized by burning or aching and is the focus of diagnostic and therapeutic techniques that can interrupt the sympathetic afferents. Conversely, interruption of the sympathetic efferents will lead to vasodilation in the area of the block and can be employed for any condition that is maintained through vasoconstriction and/or vasospasm.

Evidence/Techniques

The Sphenopalatine Ganglion Block

The sphenopalatine ganglion (also known as the sphenomaxillary ganglion, Meckel's ganglion, and the pterygopalatine ganglion) is a mixed sympathetic and

parasympathetic ganglion that has been the focus of treatments for over a century. It is triangular shaped and lies in the pterygopalatine fossa, receiving input from the preganglionic parasympathetic fibers via the greater superficial petrosal nerve (branch of the facial nerve), postganglionic sympathetic fibers from the carotid plexus via the deep petrosal nerve, and sensory afferents via the maxillary division of the trigeminal nerve. Thus, through complex and overlapping systems it supplies sympathetic tone, sensory innervation, and lacrimal gland tone of the nasal cavity, palate, major cerebral arteries, face, and orbit. It is a relatively superficial structure that can either be accessed extraorally or transnasally. Commonly accepted indications for this block are acute and chronic cluster headaches, atypical headache, trigeminal neuralgia, and herpes zoster involving the ophthalmic nerve. In a recent study by Narouze, 15 chronic cluster headache patients with positive diagnostic blocks of the sphenopalatine ganglion underwent RF ablation with significant improvement in attack frequency and intensity for up to 18 months.³⁵ Similarly, in a retrospective analysis by Sanders 60.7% of 56 patients treated with RF ablation experienced complete relief over a period of 12 to 70 months.³⁶

Stellate Ganglion Block

The stellate ganglion (cervicothoracic ganglion or inferior cervical ganglion) is the fusion between the inferior cervical ganglion and the first thoracic ganglion. It is approximately 1 cm in length and lies anterior to the transverse process of the seventh cervical vertebra and the first rib, posterior to the vertebral artery, lateral to the carotid artery, and superior to the apex of the lung. It supplies sympathetic tone to the ipsilateral upper extremity and head. The typical indications for a stellate ganglion block are complex regional pain syndrome (CRPS) (both types I and II) of the upper limb, arterial insufficiency of the upper limb (Raynaud disease, acute frostbite, scleroderma, obliterative vascular disease), herpes zoster of the face or neck, phantom limb pain, and refractory angina.³⁷ Evidence for a stellate ganglion block for CRPS has been positive, although a double-blind randomized control study has not been performed to date. In a meta-analysis by Hassantash in 2003, 110 articles with 1,528 patients who underwent either a lumbar sympathetic block or stellate ganglia sympathectomy for causalgia found that 94% responded to the interventional therapy. In a recent Cochrane review on the subject, the authors state that it should be “used cautiously in clinical practice, in carefully selected patients.”³⁸

The classic approach to a stellate ganglion block is to pierce the skin over the C6 tubercle (Chassaignac tubercle) and then advance until the needle touches down on the tubercle. The needle is then withdrawn 2 to 3 mm and contrast is injected under fluoroscopy to rule out intravascular injection. If correctly placed, the contrast should spread along the anterolateral aspect of the vertebral bodies in both posteroanterior (PA) and lateral views. Alternatively, an ultrasound-guided technique involves directing a 25-gauge needle toward the middle of the longus colli with the end point of penetration being the prevertebral fascia in the longus colli.³⁹ Horner syndrome may occur on the ipsilateral side secondary to interruption of the sympathetic fibers (it is not a reliable indicator of a successful or unsuccessful block). Although a temperature increase in the ipsilateral limb is the most often used clinical indicator, a sweat test may be a more consistent and genuine indicator of a complete block.⁴⁰

Thoracic Sympathetic Block

Thoracic sympathetic blocks are most often used for painful conditions of the chest wall including herpes zoster, postherpetic neuralgia, and phantom pain of the chest wall following mastectomy. Pain caused by tumors of the lungs, whether secondary or primary, may also yield to a thoracic sympathetic block.

The thoracic sympathetic ganglia lay lateral to the vertebral bodies and anterior to the ribs and transverse processes. The classic block technique is to enter the skin 1.5 inches lateral to the spinous process and advance perpendicular to the skin until the needle comes into contact with the transverse process. The needle is then passed inferior to the transverse process and follows the edge of the vertebral body anteriorly until the needle tip is visualized by lateral radiograph to be in the anterior third of the vertebral body. To avoid a pneumothorax, special care must be taken to hug the vertebral body medially.

Celiac/Splanchnic Nerve Block

The sympathetic chain from T5–12 gives rise to the greater, lesser, and least splanchnic nerves, which form the celiac plexus. This web of nerve fibers provide sympathetic tone to all abdominal viscera with the exception of the transverse and descending colon, rectum, and pelvic viscera.⁴¹ For this reason, it is a powerful tool for controlling abdominal pain.

Direct comparisons of unresectable pancreatic cancer patients who have undergone celiac plexus neurolysis with those who were pharmacologically managed show greatly reduced opioid need and, as a result, such reduced medication side effect profiles.^{42–45}

The story with chronic pancreatitis is not as clear, however, and both pain specialists and gastroenterologists have been plagued with a lack of clear evidence for celiac plexus blockade in this patient population.⁴⁶

The percutaneous posterior approach varies slightly for the celiac plexus versus the splanchnic nerves, but the starting point for both is lateral to the L1 vertebra. From this point the needles are passed anteriorly to either the anterolateral aspect of the T12 vertebral body (for a splanchnic block) or to 1 to 2 cm anterior to the L1 vertebra in the case of the celiac plexus blockade. The most common side effects include hypotension and diarrhea.

Lumbar Sympathetic Block

The lumbar sympathetic chain is a retroperitoneal structure that lies at the anterolateral aspect of the vertebral bodies. Its contributions include preganglionic sympathetic fibers from T10 to L2–3 and it is noted to be more medial and anterior than the thoracic chain. It has a distinct separation from the somatic nerves because it lies anterior to the aponeurosis of the psoas fascia and muscle. The left side is also posterior and lateral to the aorta and the right chain is posterior to the vena cava. It is a notably variable aspect of the sympathetic chain and is most commonly present at the inferior aspect of L2 and the superior aspect of L3.

The lumbar sympathetic block (LSB) is indicated for pelvic malignancies, chronic regional pain syndrome, peripheral vascular disease, postherpetic neuralgia, and acute herpes zoster. Its role in CRPS has been validated with improved func-

tionality and long-term improvement of pain. In one study, 29 patients with documented CRPS type I underwent LSB with local anesthetic, and 25 of 29 patients had either complete relief (45%) or partial relief (41%). Another study compared patients with a positive (pain-relieving) sympathetic block with those who did not respond to a sympathetic block. They subsequently administered weekly blocks to those who responded and found that those receiving blocks had significantly superior long-term improvement of pain and functionality.⁴⁷

The most common (“paramedian”) approach is a posterior approach in which the patient is prone and the target includes the anterolateral aspect of the L3 vertebral body with the ideal position with the needle tip in the anterior third of the vertebral body on lateral radiograph and medial to the lateral margin of the vertebra on AP radiograph.

Superior Hypogastric Plexus Block

The superior hypogastric plexus is a retroperitoneal network that is typically adjacent to the anterior aspect of the fourth lumbar vertebra body to the upper aspect of the first sacral vertebra body. It is composed of both sympathetic and parasympathetic fibers (S2–S4), which innervate the bladder, rectum, uterus, vagina, and prostate. Blocking these fibers may help to distinguish the etiology of pain as a diagnostic tool (such as distinguishing between visceral pain and referred pain from lumbar pathology), but it is most frequently employed as a neurolytic technique for cancer involving the above-listed viscera.

In a study with 28 pelvic cancer patients (cervix 20, prostate 4, testicle 1, other 3) with pain refractory to other modalities, 26 patients had pain relief of 70% or more with superior hypogastric neurolysis.⁴⁸ This was followed up by another study in which 227 patients with dull, visceral pain (from gynecological, colorectal, or genitourinary [GU] cancer) had an immediate positive response (79%) and the majority (69%) had continued pain relief at 6 months.⁴⁹ Furthermore, in a randomized trial comparing superior hypogastric block with opioid use, SHP block reduced opioid consumption and pain intensity and enhanced quality of life.⁵⁰

Ganglion Impar

The ganglion impar (ganglion of Walther) is a solitary retroperitoneal structure that supplies sympathetic input to the coccyx, rectum, anus, vulva, urethra, and vagina. It represents the cephalic tail of the sympathetic chain and is a single, fused ganglion. Indications for this block include coccygodynia and malignancies of the pelvic area.

Two techniques to block the ganglion impar are widely used, the transanococcygeal membrane technique and a transsacrococcygeal approach. In a recent prospective study involving 16 patients with chronic pelvic pain, the transsacrococcygeal approach was employed with 100% efficacy in reducing the pain by 50% with a mean time to perform a therapeutic block of 5.7 minutes.⁵¹ A similar approach was employed in a case series using only local anesthetic for treatment of coccygodynia in which a majority of the patients reported pain relief.⁵² Reig et al performed a prospective study employing RF ablation of the ganglion impar in patients with a diagnostic block with an average pain relief of 2.2 months.⁵³

■ Conclusion

Injection techniques are commonly performed in the treatment of pain by a variety of practitioners in many different contexts. Despite the growth in the numbers and types of procedures performed in recent decades, evidence to define their appropriate use has accumulated at a slower pace. Since the dawn of modern pain medicine with the publishing of Bonica's 1953 text *The Management of Pain*, the need for multidisciplinary care has been recognized. Hopefully, pressures from health care systems and other financial restraints will provide increased incentives to collect meaningful data to define the appropriate role of these procedures in modern pain practice.

Editor's Comments

Anesthesiologists have been instrumental in the adoption of improved standards of acute and chronic pain care in hospitals and the outpatient environment. Our colleagues also implant neurostimulation systems in the spinal and peripheral nerves, as well as intrathecal drug administration systems. This chapter highlights the common procedures that are performed by anesthesiologists in patients with chronic pain. Dr. Stacey and associates have given us concise reviews of epidural steroid injections (ESIs) and medial branch blocks, and a more detailed treatment of various lytic autonomic blocks.

ESIs are controversial, but remain the mainstay of many interventional pain centers. The data are complex but overall indicate that ESIs:

- Are best for acute radiculitis
- Should be immediately combined with a physical rehabilitative program to optimize therapeutic benefit
- Do not consistently help back pain
- Are not useful in cases of spinal stenosis

Medial branch blocks are more likely to be of some benefit for neck pain, but the palliative effect is measured in months. Unfortunately, neck pain is usually a chronic condition, so the pain inevitably returns. Good outcomes with lumbar medial branch blocks are uncommon, and usually short-lived. I do not see the latter procedure performed with any regularity, and that may be a consequence of a lack of enthusiasm for the procedure by many insurance companies.

Autonomic blocks are used in a variety of conditions, and range from sympathetic (sphenopalatine, stellate, thoracic, celiac/splanchnic, lumbar, ganglion impar) to mixed sympathetic/parasympathetic (hypogastric). The efficacy of these blocks may be attributable to the sympatholytic effect or, more likely, the disruption of nociceptive C fibers that course through these ganglia en passant. Over the past decade, the concept of "sympathetically maintained pain" has been challenged, and it is difficult to gain perspective on how often these procedures are performed. I believe the treatment of complex regional pain syndrome (CRPS), either type I or type II (causalgia), using autonomic blocks has fallen out of favor, as evidenced by the cautious conclusion of the Cochrane review cited by the authors.

In contrast to "sympathetic blocks," celiac blocks are still used for cancer pain of the abdominal viscera and have a track record of efficacy. Similarly, superior hypogastric or ganglion impar blocks can help pelvic pain related to malignancy.

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Section II

Pain Surgery Techniques and Procedures

Section II.A

Spinal Cord Stimulation

3

Spinal Cord Stimulation: Patient Selection

Richard B. North and Giancarlo Barolat

Soon after the introduction of percutaneous electrodes and a mere 5 years after Shealy had initiated the therapy,¹ Hosobuchi et al. conducted the first spinal cord stimulation (SCS) screening trial.² Patient selection was already an issue: Clinicians needed to understand why SCS successfully reduced pain in some patients but not in others. Today patient selection for SCS remains a multistage process that begins with a referring physician and ends with a specialist deciding whether to offer a screening trial and then (in consultation with the patient) whether to proceed with SCS implantation after the trial. All referring physicians should understand which patients might be candidates for an SCS screening trial and how the specialist will interpret the trial's outcome.

SCS screening trials are usually minimally invasive, technically straightforward percutaneous procedures. For patients with complicated (e.g., postsurgical) anatomy, however, a plate/paddle electrode might be required. (Details about the techniques and equipment involved in an SCS screening trial are provided elsewhere in this book.) SCS screening trials not only help define the optimal position of the electrode and patterns of stimulation, they also emulate the chronic therapy more accurately than do other types of prognostic trials for other modalities. The results of a screening trial offer the best prognosis of SCS outcome.

■ Indications

SCS is offered as a treatment for chronic neuropathic pain, ischemic conditions (to improve blood flow as well as for pain control), and conditions that cause visceral pain. SCS patients will ideally obtain a specific diagnosis sufficient to explain the type of pain they are suffering. Pain, however, is often mixed: Primarily neuropathic pain might have nociceptive components (signaling actual or impending tissue damage) and/or ischemic components, and primarily ischemic pain might have neuropathic and/or nociceptive components.

SCS for Neuropathic Pain

In the United States, treatment of chronic neuropathic pain is the most common (and most accepted) indication for SCS. Neuropathic pain responds better to SCS than does nociceptive pain, and it is technically easier to overlap the lower extremities with stimulation paresthesia than the low back; thus, it is easier to treat radicular or radiating neuropathic pain than axial low back pain, which commonly has nociceptive components. With the development of programmable multicontact devices and the refinement of implantation and programming techniques, the selection criteria for an SCS screening trial now include patients with predominantly axial low back pain.³

SCS is used to treat neuropathic pain caused by, for example, failed back surgery syndrome,^{4,5} a spinal cord lesion with well-circumscribed segmental pain,⁶ peripheral nerve injury,⁷ phantom limb/postamputation syndrome,⁸ postherpetic neuralgia,⁹ and complex regional pain syndrome.¹⁰

SCS for Ischemic Conditions

SCS is used to treat conditions that cause ischemic pain, such as refractory angina pectoris¹¹ (including syndrome X¹²), peripheral arterial occlusive disease,¹³ frostbite,¹⁴ Raynaud syndrome,¹⁵ and diabetic neuropathy.¹⁶ SCS can have a positive effect not only on ischemic pain but also on the underlying microcirculation. Whereas SCS might be considered a front-line therapy in patients with Raynaud syndrome or frostbite, it is generally reserved as a last option for those with refractory angina pectoris or peripheral arterial occlusive disease. Thus, the outcome criteria used to document the impact of SCS on ischemia extend beyond pain control to include patient survival, limb salvage, and microcirculation improvement.¹⁷

SCS for Visceral Disease

SCS is also being used to treat several diseases that cause visceral dysfunction and pain—for example, interstitial cystitis,¹⁸ pancreatitis,¹⁹ urinary urgency and frequency,²⁰ pelvic pain,^{21,22} vulvodynia,²³ and abdominal pain.²⁴ In these cases, SCS can have a positive impact on the underlying disease as well as its symptoms.

■ Techniques

Techniques of Patient Selection for an SCS Screening Trial

A patient with an appropriate diagnosis as noted above should be considered for SCS after less or equally invasive alternative therapies have failed to provide relief or have provided relief only with intolerable side effects. In most cases, patients won't be referred for SCS until such therapies have failed or are contraindicated. For patients with failed back surgery syndrome, however, unless a repeat lumbosacral procedure is required to address a neurologic deficit that might become permanent, it is appropriate to offer an SCS screening trial rather than a repeat lumbosacral surgical procedure, even in the presence of a potentially surgically remediable condition, such as disk herniation.⁵

Information to determine a patient's suitability for a screening trial is gathered from several sources:

- The history, which must include information about pain location and intensity, therapies that have failed, current medications, allergies, and comorbid pain conditions. If the patient had prior lumbosacral surgical procedures, the operative records will provide important information.
- A physical examination, which could reveal abnormalities that require surgical intervention before or instead of SCS and should provide information on functional aspects of the patient's pain.

- Imaging studies, such as magnetic resonance imaging (MRI), which will reveal the presence of stenosis, disk herniation, or another anatomic abnormality that could increase the procedural risk of SCS. An MRI will also document the depth of dorsal cerebrospinal fluid and the position of the spinal cord, and this information will contribute to the selection of the SCS equipment and implantation site.
- A psychological evaluation, which should uncover any major untreated psychiatric or personality disorder, issue of secondary gain that could confound SCS outcomes, serious drug habituation problem, or abnormal illness behavior. SCS patients must also demonstrate the cognitive and physical abilities necessary to operate the programming equipment.

All of these factors must be addressed prior to a screening trial using a laminectomy procedure or implantation for chronic therapy.

Additional selection criteria for patients with angina or syndrome X include:

- New York Heart Association Class III or IV angina
- Revascularization therapy contraindicated
- Demonstrated reversible myocardial ischemia
- No recent (< 6 mo) acute myocardial infarction
- Absence of comorbid heart disease (e.g., peri- or myocarditis)

Additional selection criteria for patients with peripheral arterial occlusive disease are:

- Severe pain at rest
- Reconstructive vascular surgery contraindicated
- Any ischemic ulcer < 3 cm in diameter
- Any dry gangrene
- TcPO₂ between 10 and 30 mm Hg²⁵

The following are potential contraindications to SCS, even before a screening trial:

- Age under 18 years (Per device labeling, the safety of SCS has not been established in children; however, the authors have implanted patients under age 18 and have not observed any untoward side effects or complications.)
- Unresolved major psychiatric comorbidity
- Unresolved likelihood of secondary gain
- Active and untreated substance abuse disorder
- Inconsistent or abnormal findings in the history, pain description and rating, diagnostic studies, or physical examination (such as a predominance of non-organic signs, e.g., Waddell sign) are nonspecific.²⁶ Symptoms or signs that seem odd or uncommon should be interpreted with caution.
- Alternative therapies with a risk/benefit ratio comparable to that of SCS have not been ruled out.
- Occupational risk (e.g., the patient works on a ladder)
- Local or systemic infection (Chronic or ongoing septicemia is an absolute contraindication.)

- Foreseeable need for an MRI, unless MRI safe systems are utilized
- Potentially disabling neurologic deficit attributable to a surgically correctible problem (e.g., nerve compression)
- Presence of a demand pacemaker (but see the 2003 study²⁷ in which thoracic SCS was safely used in angina patients with a pacemaker)
- Presence of a cardioverter defibrillator (but see the 1998 report of Monahan et al²⁸)
- Presence of another major comorbid chronic pain syndrome (This generally would be a contraindication for participation in an SCS clinical trial because it could confuse results.)
- Anticoagulant or antiplatelet therapy that cannot be reversed temporarily to allow SCS implantation
- Ulcers close to implantation sites
- Pregnancy (but SCS might be safer than other forms of pain therapy²⁹)
- Brief life expectancy (but SCS with an external pulse generator might be useful)

Possible Prognostic Factors

Several investigators have attempted to identify prognostic factors of success by statistical analysis of patient-specific characteristics (age,^{30,31} gender,³² number of prior surgical procedures, time since last surgical procedure, presence of an issue of secondary gain, etc.) or pain-specific characteristics³³ as well as by the technical aspects of treatment (type of electrodes^{34–36} or pulse generators³⁷). Whereas improved outcomes are well documented for certain technical improvements, such as programmable multicontact devices, the most useful clinical prognostic factor is the degree to which pain is relieved during the SCS screening trial.

Techniques of an SCS Screening Trial

A screening trial in a patient with the most common indication for SCS, failed back surgery syndrome, involves placement of a low thoracic electrode, and so there is usually no impediment to percutaneous placement at the thoracolumbar junction. In some cases, as noted above, electrodes must be placed by an open surgical technique because the postoperative changes (e.g., posterior instrumentation or laminectomy defect) involve the intended electrode level(s). Other indications for SCS, which might not involve spinal pathology, are likewise generally amenable to a percutaneous trial. The equipment involved and implantation techniques are detailed later in this book. Two technical decisions, however, merit discussion here.

First, during the screening trial, a percutaneous SCS electrode may be either anchored subcutaneously and connected with temporary percutaneous extension wires, or allowed to emerge from its insertion point and secured only to the skin. Advantages of the first approach (anchored, with tunneled extension) include the following:

1. Eliminating the expense of a second electrode should the patient pass the trial
2. Allowing the trial to be conducted with the definitive electrode in the definitive position

Advantages of the second approach (strictly percutaneous, strictly temporary, with a second stage only if the trial is successful) include the following:

1. Eliminating the need for two trips to the operating room, with its associated expense, because no incision is required and no foreign body is implanted permanently. The operating room is required only if a patient receives a permanent implant. An anchored, tunneled electrode always requires a second trip to the operating room, if only to remove the electrode after an unsuccessful trial.
2. Providing the opportunity to improve the position of the temporary electrode, which was placed in a naïve patient, with a permanent electrode placed in a now experienced patient
3. Providing the opportunity to select the most appropriate permanent electrode based upon the results of the trial (e.g., a paddle electrode in a patient whose otherwise successful percutaneous trial was confounded by stimulation-evoked midback pain, attributable to recruitment of small pain fibers in ligamentum flavum)³⁷
4. Avoiding the pain associated with the incision, anchoring, and tunneling. Such pain might require analgesics and confound interpretation of the trial. In some instances, a “tunnelled trial” is performed with a paddle lead. In this instance, if the trial is successful, the lead is left in place and the IPG is inserted in the second procedure. Such a trial is indicated in situations where placement of a percutaneous lead is not possible or when the percutaneous lead cannot be steered to the correct location.

■ Outcomes

In our experience 80 to 90% of patients referred specifically for SCS pass the initial records review (before we schedule an appointment). Once seen, 80 to 90% progress to a screening trial, and 65 to 85% pass the screening trial.

What Constitutes a Successful Screening Trial?

Interpreting the results of an SCS screening trial should be straightforward. The purpose of the trial is to establish that pain relief will be adequate and satisfactory to the patient (who must prefer the feeling of paresthesia to the pain and must be content to live with an implanted device). SCS is not expected to alleviate pain completely (although that outcome can occur), and patients must understand this. The patient must also understand that we do not believe that SCS reduces nociceptive pain, including pain from ulcers or gangrene in patients with peripheral arterial occlusive disease.

Technical success (overlapping pain with comfortable paresthesia) is necessary (but not sufficient) to achieve clinical success (reduction in pain). Although some investigators report the use of subperception stimulation,³⁸ we must await more evidence before we can determine if this technique is successful. In the meantime,

the technical goal of a screening trial is to achieve as much pain/paresthesia coverage as possible with stimulation at a level that is above perception and below that which elicits discomfort and/or involuntary movement.

Clinical success occurs when a patient reports at least 50% reduction in pain by standard rating methods and demonstrates improved or stable analgesic requirements despite provocative activities. (Some investigators, however, consider this percentage of relief to be too stringent.³⁹) Patients with peripheral arterial occlusive disease should also demonstrate a clear increase in TcPO₂ or in some other objective indicator of microflow improvement. If the clinical results of a technically adequate trial are not convincing to the patient and the physician, prolonging the trial period can help to clarify the situation.

In our experience, 65 to 85% of patients pass the screening trial, and nearly all patients who pass the screening trial receive an implanted system for chronic use.

Repeat Trials

In some cases, patients experience uncomfortable paresthesia outside the bounds of the target area; this situation can warrant a repeat trial, sometimes with an insulated electrode that can prevent this side effect.⁴⁰ A repeat trial is also in order when the disappearance of initial clinical success occurs with technical problems (e.g., electrode migration). Sometimes a trial must be aborted because of an adverse, temporary biological response (epidural hematoma, infection) that must be resolved. After resolution, the trial can be repeated.

What Causes Trial Failure?

An SCS screening trial fails when it is impossible to cover the pain with comfortable paresthesia (technical failure), when pain–paresthesia overlap is technically successful but does not reduce pain (clinical failure), or if a psychological complication (e.g., the patient develops an aversion to an implanted device) occurs (patient-related failure).

■ Conclusion

To obtain a positive outcome with SCS treatment, we must offer the therapy to the correct patient with the correct indication. In addition, we must use the correct equipment and the correct technique; this means that the implanting physician must have the proper education and experience. Patient selection for an SCS screening trial can be liberal if interpreted conservatively, and it can be considered before every other therapy has been exhausted.

Patient selection for an SCS screening trial conducted as part of a clinical trial must be more rigid to avoid introducing unnecessary uncontrollable factors into the trial.

Several categories of independent variables are pertinent to patient selection and have demonstrable associations with SCS outcome:

1. Organic disease/diagnosis
 - a. Primary
 - b. Iatrogenic

2. Psychological status
 - a. Premorbid
 - b. Secondary
3. Treatment
 - a. Choice of SCS; choice of device
 - b. Trial methodology and technical success
 - c. Trial interpretation

All of these variables should be considered in the selection of patients for device implantation.

Editor's Comments

As mentioned in this chapter, patient selection is of paramount importance in utilizing spinal cord stimulation (SCS) for the treatment of chronic medically intractable pain. Drs. North and Barolat are both masters of this technique, and both have contributed substantially to our understanding of the application of this procedure. They have presented their paradigm for patient selection in a clear and succinct manner, and I concur with almost all aspects of their report.

One area that remains difficult is the treatment of low back pain with SCS. As they note, back pain probably has a major nociceptive component, and therefore is not *a priori* as responsive to SCS as is neuropathic extremity pain. Furthermore, as alluded to in the chapter, production of paresthesias in the midline back—a must for successful pain relief—is more problematic than it is in the dermatomes of the lower extremities.

The authors of this chapter believe that patients with “predominant axial low back pain” should be considered candidates for SCS. Based on my experience, I am not convinced. In the condition that is clearly the most common indication for SCS, the failed back surgery syndrome (FBSS), I have experienced initial success with both back and leg pain, only to be disappointed with the discontinuation of back pain relief in longer term follow-up. As a result, I have avoided this indication for SCS.

As has been demonstrated previously by Dr. North,⁵ in prospective trials, SCS almost certainly produces superior, and more cost-effective, outcomes than repeated major back surgery for FBSS. It continues to amaze me that we routinely see patients subjected to multiple major spinal surgeries, for which the outcomes are unproven by almost any measure, while the application of SCS in these same patients is often considered exceptional, even “experimental,” by insurance or worker’s compensation providers. The production of further evidence of the superiority of SCS over repeated spine surgery remains a challenge for the specialty of pain surgery. However, there is almost no other question in our field that can potentially have as much impact on the quality and cost of care.

I am gratified that the authors have included psychological evaluation in their screening procedures. This has been inconsistent as an area of practice, and in the reported literature on SCS. At the very least, it is important to document aspects of the patient’s history that might confound the outcome of SCS, including major depression, a strong tendency to convert psychological stress to physical symptoms (somatization), prior history of drug abuse, or major secondary gain related to the pain problem. The last motivation may be relevant in cases in which a worker’s compensation claim is being actively adjudicated. In fact, active litigation related to the pain condition is probably a substantial contraindication to proceeding with an SCS trial.

Overall, setting goals for SCS and the establishment of reasonable expectations are of great importance before one sets about to conduct a trial of SCS. Despite these caveats, SCS remains a unique tool for the relief of chronic pain, in that it is both testable and reversible. It is at once a venerable technique and a target for further study and refinement.

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4

Spinal Cord Stimulation: Equipment and Implantation Techniques

Giancarlo Barolat and Richard B. North

Spinal cord stimulation (SCS), a reversible neuromodulation technique, is one of the safest and most effective procedures available for the long-term management of certain chronic pain conditions.^{1–11} Even though a seemingly straightforward procedure, SCS implantation is technically demanding and requires care in planning and execution. The implanter has to be fastidious about the correct positioning of the electrode(s), in the longitudinal and in the transverse directions^{12,13}; about the placement of the implanted pulse generator (IPG); about the location of the subcutaneous leads; and about the system connections. If any one of these factors is not optimal, the effectiveness of the whole procedure is jeopardized. This chapter discusses SCS equipment and some technical details of system implantation.

■ Equipment

SCS equipment includes electrodes (sometimes known as “leads,” a term that includes the wires that are part of the electrode assembly), IPG, external controller, and charger. Radiofrequency receivers, although extensively used in the past and still used by some patients, are no longer being produced.

Electrodes

Electrodes (**Figs. 4.1** and **4.2**) can be classified according to their shape (cylindrical or plate/paddle) or implantation technique (percutaneous or laminectomy). The recently introduced St. Jude Medical S-Series electrode (St. Jude Neuromodulation, Austin, TX, USA), however, has blurred this distinction because it is a paddle electrode that can be implanted using a percutaneous ovoid catheter (Epiducer).¹⁴

Percutaneous electrodes can be used for a screening trial or implanted for chronic use. Most percutaneous electrodes are either quadripolar or octapolar. One 16-contact percutaneous electrode, the Infinion (Boston Scientific, Valencia, CA, USA), is available. Some electrodes connect directly to the IPG; others connect through an intermediate splitter or subcutaneous extension cable.

For the screening trial, a percutaneous electrode may be inserted in a fluoroscopy room and secured only at the needle insertion site; such an electrode is easily removed at the bedside or in the office. Alternatively, a percutaneous electrode may be inserted in an operating room and anchored subcutaneously. This method has the potential advantages of (1) saving the cost of a second electrode

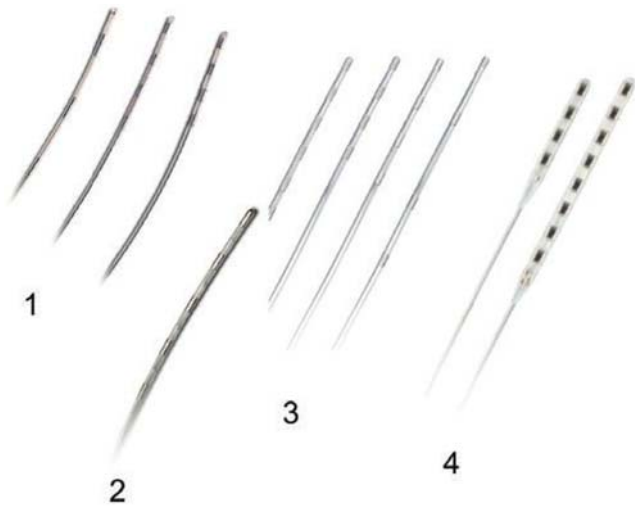


Fig. 4.1 Percutaneously inserted electrodes (not in scale). 1–3, cylindrical electrodes; 4, St. Jude S-Line percutaneously insertable paddle electrodes.



Fig. 4.2 Paddle electrodes (not in scale).

for chronic use and (2) ensuring that the permanent configuration reproduces a successful temporary one. Corresponding disadvantages are (1) the cost of a trip to the operating room for insertion; (2) the potential cost of a trip to the operating room for removal after an unsuccessful trial; (3) accepting the electrode position achieved in a naïve patient, rather than taking advantage of an opportunity to improve it after the patient has experienced trial stimulation; (4) the possibility that significant postoperative pain from the incision and anchoring procedure might confuse interpretation of trial results; (5) the possibility of creating a bias toward a positive trial; and (6) greater risk that an infection introduced by the temporary configuration will persist and involve the pulse generator.

Placement of percutaneous electrodes must be performed under fluoroscopic guidance, which requires personnel to wear heavy shielded garments and potentially exposes the patient and the implanting physician and staff to nonnegligible levels of radiation. The percutaneous approach is appealing, however, because it allows electrode insertion without muscle dissection or removal of bone tissue. In addition, percutaneous electrodes can be advanced through several segments in the epidural space to allow testing of several spinal cord levels. Multiple parallel percutaneous electrodes can be used to increase the number and variety of stimulation configurations that are tested. In the United States most implanters insert temporary electrodes for the screening trial, but in Europe most screening trials take place with electrodes implanted and anchored as if for chronic use.

Paddle electrodes are usually implanted for chronic use but can also be used for a screening trial (and can be removed or left in place if the trial is not a success). The simplest paddle electrode is quadripolar (e.g., the Medtronic [Minneapolis, MN, USA] Resume or Resume-TL, the St. Jude Lamitrode), with four contacts arranged linearly on a single paddle. Electrodes with 8 or 16 contacts increase the potential for stimulating multiple targets. These electrodes include the Specify 3998 and the Specify 2x8 (both Medtronic), the Lamitrode 44 and 88 (both St. Jude Neuromodulation), and the Artisan (Boston Scientific). These electrodes comprise two columns of narrowly spaced contacts. When centered symmetrically on the physiologic midline, the electrodes allow elective stimulation on either side or bilaterally.² Coupled with a “dual channel” control system, these electrodes allow great flexibility of stimulation. Some paddle electrodes have more than two columns of contacts, such as the St. Jude Penta (five columns) and the Medtronic Specify 3999 (three columns).

Implantation of paddle electrodes requires surgical placement under direct vision. The amount of actual bone removed can be minimal; in the cervical area, often no bone removal is necessary. Most paddle lead implants can be done through a small (1- to 1.5-inch) skin incision. By advancing the electrode in a cephalad or caudal direction, one can explore at least four spinal levels in the thoracic spine and four or five levels in the cervical spine. Multiple arrays or different electrode configurations also can be constructed with laminectomy electrodes.

The main advantages of paddle electrodes are their insulated dorsal surface (which ensures that all the stimulating current is delivered to the intraspinal structure and thus can reduce the incidence of unwanted or uncomfortable stimulation), the inherent stability of each column of contacts with respect to the others, and their inherently greater stability once encapsulated in the dorsal epidural space, which potentially reduces their propensity to migrate, compared with inadequately secured percutaneous electrodes.

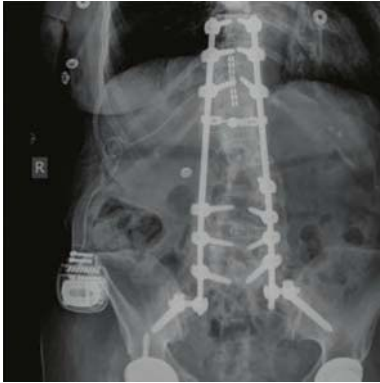


Fig. 4.3 Thoracic paddle electrode placed in previously instrumented thoracic spine.

For low thoracic electrode placement for failed back surgery syndrome, which is the most common clinical application of SCS, the pattern of stimulation-induced paresthesia provided by insulated paddle electrodes seems to be superior to that produced by percutaneous electrodes.¹⁵ In a randomized, controlled trial, the technical performance of laminectomy electrodes significantly exceeded that of percutaneous electrodes^{16,17}; concordance of stimulation paresthesia with pain was statistically better for laminectomy electrodes, and power requirements were lower by half.

Some situations clearly indicate one or the other of these two methods (i.e., a percutaneous system for trial electrode placement in a fluoroscopy suite,

or a paddle electrode for patients with a history of certain types of spine surgery) (Figs. 4.3 and 4.4). Otherwise, the choice of trial electrode is dictated by individual preferences and practice patterns. A skilled implanter usually can achieve a similar stimulation matrix with either a laminectomy or percutaneous electrode(s). The

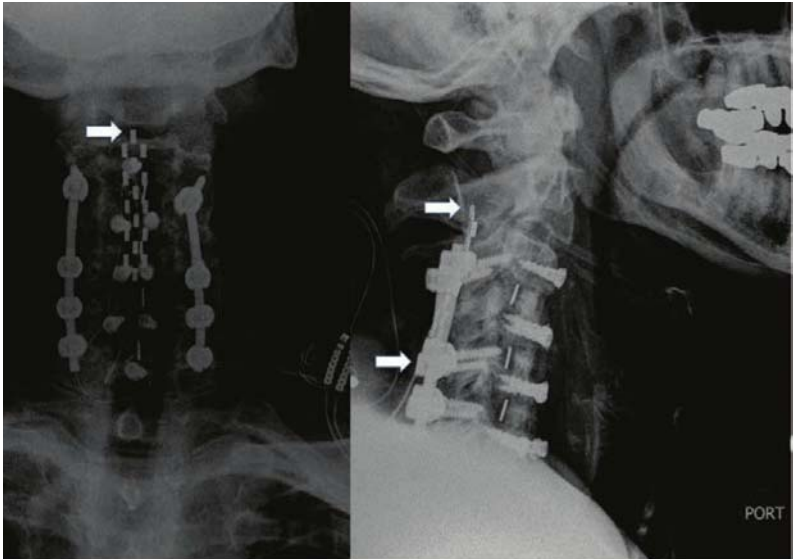


Fig. 4.4 Cervical paddle electrode placed in previously instrumented cervical spine.

choice of permanent electrode can be dictated by the results of the trial (e.g., a paddle electrode to mitigate stimulation-evoked pain, attributable to recruitment of small fibers in ligamentum flavum,¹⁶ or to improve low back coverage¹⁷).

Implanted Pulse Generator

Stimulation is delivered as charge-balanced pulses from an IPG (**Fig. 4.5**) powered by a battery through lead(s) to electrode contacts in the epidural space. Some SCS systems contain a nonrechargeable lithium battery; others contain a transcutaneously rechargeable battery. The introduction of the rechargeable battery has allowed manufacturers to reduce the size of the IPG while increasing its service life.

IPGs are activated and controlled by telemetry to and from an external control unit, or by a small external magnet, through the intact overlying skin. Stimulation is delivered at rates up to 10,000 Hz, pulse widths up to 1 ms, and amplitudes up to about 25 mA, adjustable in small increments. The life span of the battery varies with daily patterns of use and with stimulation parameters and number of active contacts. Most patients can expect, with conservative to average use, that a nonrechargeable battery will last 2 to 5 years. A rechargeable battery will have a much longer life, at least 9 years. The Medtronic rechargeable system is unique in that it is designed to shut off and require replacement at 9 years.

Medtronic's RestoreSensor IPG is the first neurostimulator to adjust stimulation automatically as the patient changes position. This IPG uses AdaptiveStim



Fig. 4.5 Implantable pulse generators (not in scale). Precision images courtesy of Boston Scientific. © 2014 Boston Scientific Corporation or its affiliates. All rights reserved. RestoreSensor SureScan MRI neurostimulator with leads reprinted with the permission of Medtronic, Inc. © 2013. Eon Mini rechargeable IPG (implantable pulse generator) image courtesy of St. Jude Medical. Eon Mini and St. Jude Medical are trademarks of St. Jude Medical, 2014. All rights reserved.

technology, which automatically correlates changes in body position with the required level of stimulation. AdaptiveStim also records data regarding patient activity that clinicians can use to assess, evaluate, and optimize a patient's neurostimulation experience.

The Boston Scientific system employs multiple independent current control (each contact has a dedicated power source), which could result in a more discrete distribution of the current in the neural structures than is available with the other IPGs. The Boston Scientific Spectra pulse generator is the only one offering 32 independent stimulation channels.

Nevro (Menlo Park, CA, USA) produces an IPG that can deliver stimulation up to 10,000 Hz. As of this writing, it has not received FDA approval for use in the United States.

■ Operative Technique

Intraoperative Anesthetic Management and Testing

Electrodes can be implanted with the patient under monitored anesthesia (local anesthetic and intravenous sedation) or under general anesthesia. Unlike SCS applied for motor disorders or for ischemic conditions, conventional SCS for pain requires that stimulation-induced paresthesia cover a patient's area(s) of pain. Thus, it is logical that testing an awake and cooperative patient yields the best results.

When implantation is performed using general anesthesia, one can rely only on radiologic position and on evoked motor or sensory responses, and cannot obtain specific information about the distribution of the paresthesia or about discomfort at or near the therapeutic threshold. It is one author's (G.B.) preference, therefore, even when the implantation of the electrode is performed under general anesthesia, to rouse the patient during the procedure to ensure proper electrode placement; intraoperative wake-up is safely performed in over 90% of his cases. The other author (R.N.) prefers local anesthesia, supplemented by brief sedation as necessary, and finds it applicable to over 90% of cases. Thus, both authors prefer to engage in meaningful conversation with an awake and cooperative patient during the intraoperative testing phase of a screening trial or implantation for chronic use. Of course, the safety of the procedure is paramount, and anesthetic technique has to be determined on an individual basis.

When conducting intraoperative testing, the implanting surgeon must be aware of the different responses that stimulation of the various intraspinal structures will elicit. The strategy for electrode placement in both the transverse and the longitudinal directions varies according to the pain topography and intraoperative-elicited responses.^{8,9}

If the implantation is performed with the patient under general anesthesia, one must rely on three factors for electrode placement:

1. *Intraoperative X-ray.* Intraoperative localization, either by plain radiograph or fluoroscopy, can be used to locate the spine level and lateralization of the electrode. Correlation between right and left electrode locations and lateralization of paresthesia is imperfect, however, because the physiologic midline of the spinal cord does not always correspond with the radiologic midline.¹⁰

Even when the image shows a centrally placed electrode, stimulation might not elicit bilateral, symmetric sensory (or motor) effects.

2. **Motor testing.** Stimulation is delivered through the implanted epidural electrode at a rate of 2 to 5 Hz, and elicited motor contractions are observed or recorded through previously inserted needle electrodes in the desired muscle groups. If stimulation through the epidural electrode triggers motor contractions in the painful extremity(ies), paresthesia will likely also be perceived there with a therapeutic level of stimulation. This principle is usually reliable in the cervical area, but direct patient reports of sensory effects are more useful in the low thoracic spine. In one author's (G.B.) experience (since he routinely performs evoked motor potentials followed by patient wake-up) in about 50% of instances the information provided by the motor potentials is not satisfactory and the lead(s) must be repositioned.
3. **Evoked potentials.** Some researchers have advocated the use of somatosensory evoked potentials or compound action potentials to localize the position of the electrode.^{18,19} We have used these techniques in a minority of cases, in which direct patient feedback is not feasible. Like motor stimulation, they represent a proxy for direct patient reports, which remain the "gold standard." As a general rule, implantation with the patient under general anesthesia with no intraoperative wake-up test cannot provide the same level of accuracy of placement as implantation with sensory feedback from the awake patient, who ultimately will be the judge.

Percutaneous Electrode Placement

Patient Positioning

Percutaneous electrode placement is routinely performed with the patient in a comfortable prone position on a padded fluoroscopy table. A pillow underneath the abdomen will create some degree of kyphosis, which might facilitate electrode insertion. It is important to ensure that the patient's trunk (for thoracolumbar placement) or neck (for cervical placement) is in a neutral position without rotation or twisting.

Occasionally, if a patient cannot tolerate the prone position, the procedure can be performed in the lateral decubitus position, but this makes intraoperative fluoroscopic assessment more difficult, and electrode position with respect to the midline might not be maintained postoperatively. In some centers, the procedure is routinely performed with the patient seated, which emulates the position in which the device will likely be most used, might add to the patient's comfort during insertion, and might increase the implanter's ability to obtain thoracolumbar kyphosis, which facilitates insertion of the needle in the epidural space.

Percutaneous Electrode Insertion

The level of electrode insertion is determined by several factors. Ideally, several centimeters of the electrode body should lie within the epidural space to minimize dislodgment, and thus insertion should start at least two spine segments below the desired target. Another consideration is choosing a relatively kyphotic level, to facilitate achieving a shallow angle of approach to the epidural space. Yet another consideration is choosing a level that will minimize the risk of injury at the cervical or

lumbar cord enlargement. Commonly chosen levels, therefore, are at or just below the cervicothoracic and thoracolumbar junctions.

The fluoroscopy equipment must be ready to function in both the anteroposterior and lateral planes at the time of Tuohy needle insertion. The Tuohy needle is inserted at as shallow an angle (as nearly parallel to the spinal canal) as possible. In the thoracic area, this can be accomplished with either a midline or paramedian approach; in the upper lumbar area, a paramedian approach is required. A shallow angle greatly facilitates subsequent electrode insertion and control of the electrode in the epidural space. A paramedian insertion allows such a shallow angle of needle placement and avoids placing the electrode between two adjacent spinous processes. (Extension of the spine can pinch a lead between two spinous processes; this can lead to fatigue fracture.)

When the epidural space is satisfactorily identified, the electrode is gently inserted and steered under anteroposterior (and, as necessary, lateral) fluoroscopic guidance, with test stimulation at representative contact combinations along its length as it is advanced, level by level, to establish its position with respect to the physiologic midline and the target segments. Once the desired electrode location in the epidural space has been established, the emerging lead must be secured for stability. A temporary percutaneous electrode is commonly secured by a skin suture; special devices and techniques for permanent anchorage via an incision have evolved to mitigate the problem of postoperative migration.²⁰

Paddle Electrode Placement

Two basic positions can be used: prone or semilateral. The prone position allows a straightforward appreciation of spatial relations and is the one generally used for posterior surgical approaches to the spine. The prone position is used for implantation under general orotracheal anesthesia without intraoperative patient wake-up. The potential difficulty in airway management, however, limits the use of generous intravenous sedation to supplement local anesthesia in the prone patient.

In the semilateral position, the patient lies in a “park bench” position, exposing the spine as well as the flank, abdomen, or buttock for the IPG implant. In this position, airway management is easier and thus safer than in the prone position, and the anesthesiologist feels more comfortable in administering general anesthesia (usually with a laryngeal mask) and then waking up and extubating the patient for intraoperative testing. Deep sedation is also administered more safely in this position than in the prone position. One must be aware, however, that because of variable rotation of the body, three-dimensional visualization of the operated structures may be less intuitive. This problem is compounded in the cervical area where rotation and flexion/extension of the spine occur.

Based on his experience with more than 8,000 implanted paddle electrodes, one of the authors (G.B.) prefers the semilateral position; the other (R.N.) prefers the prone position, based on limited experience with laminectomy electrodes and a large amount of experience with percutaneous electrodes under local anesthesia, which he finds applicable to the majority of permanent SCS implants. Individual patient considerations and practitioner experiences and preferences should guide these choices.

In the planning phase of the procedure, the implanting surgeon must be aware of the varying angulation of the spinous processes at different spinal lev-

els and of the correlation between the various spinal levels and the patterns of stimulation-induced paresthesia.¹² All three manufacturers of paddle electrodes recommend imaging (typically MRI) before implantation to assess the diameter of the spinal canal; this improves patient safety.²¹ Some (author R.N. included) prefer imaging even before percutaneous electrode placement. Prior to incision or needle placement, a radiographic or fluoroscopic image is taken in the operating room with metallic markers placed on the skin at the level of the planned incision. This allows precise marking of the entry level.

In a typical case, with ample spinal canal diameter to accommodate the paddle electrode safely, it is introduced beneath the intact neural arch into the dorsal epidural space through a minimal exposure. Subperiosteal dissection usually can be limited to the upper half of the spinous process inferior to the desired interlaminar space and to at least the inferior two-thirds of the spinous process superior to it. Parts of the superior spinous process can be incrementally removed until the ligamentum flavum is exposed. Insertion of a paddle lead can also be performed without removing the spinous process nor the supraspinous ligament. This preserves the structure of the spine and minimizes postoperative pain and spine instability. Following removal of the ligamentum flavum, and a limited laminectomy as necessary to accommodate the electrode(s) safely, the dorsal epidural space is dissected bluntly, and then the electrode is inserted at a shallow angle. Cases with significant stenosis at the planned level of electrode implantation can be addressed by decompressive laminectomy. After placement of the electrode, the emerging lead can be anchored in the same fashion as for percutaneous leads.

■ Implantation of the IPG

The position of the IPG is crucial and must be comfortable for the patient. The most common placement sites include the abdomen, the fat pad in the posterior iliac area, the infraclavicular area, and the lateral chest wall area. In most cases, the IPG is placed in the subcutaneous tissue. In thin patients, it can be useful to place the IPG under the fascia. The unit can be secured to the underlying tissues with nonabsorbable sutures. In patients with abundant adipose tissue or loose subcutaneous tissue, the IPG may be placed in a Dacron pouch to reduce the chance of unwanted movement. This pouch can be secured with multiple sutures to both the deep and the superficial subcutaneous layers. This maneuver will minimize, but not completely eliminate, the risk of migration and tilting. The subcutaneous pocket is made with either blunt (fingers, Kelly clamps) or sharp (scissors, cutting coagulator) dissection. The pocket should be only slightly larger than the IPG to reduce the likelihood of migration. Ideally, the whole unit (including the connectors) will lie completely within the pocket and will not cross underneath the skin incision.

■ Conclusion

Refinements in surgical technique, along with device development, continue to improve the safety, efficacy, and reliability of SCS, as is appropriate for neuromodulation, which is intended as reversible therapy. SCS is increasingly the treatment of choice for complex, refractory chronic pain conditions.

Editor's Comments

This chapter is a companion piece with Chapter 3. Two of the most notable spinal cord stimulator (SCS) implanters, Dr. Rick North and Dr. Giancarlo Barolat, have shared with us their combined wisdom on the implantation of SCS devices. Each surgeon elucidates the “ideal” method for implantation, and this is a product of experience, the nature of the unique patient population, and the clinical setting. I have my own preferences, but the differences are fairly trivial.

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Section II.B

Peripheral Nerve Stimulation

5

Peripheral Nerve Stimulation

Viraat Harsh and Ashwin Viswanathan

In 1967 Patrick Wall and William Sweet¹ described the first experience with the use of peripheral nerve stimulation (PNS). Their initial explorations into PNS were based upon one of the predictions of the “gate control” theory of pain as delineated by Ronald Melzack and Patrick Wall 2 years prior.² Specifically, they postulated that stimulation of large-diameter cutaneous afferent fibers can lead to decreased pain perception. With this as the basis, Wall and Sweet inserted needle electrodes into their own infraorbital nerves and were able to produce paresthesias in the sensory distribution of the nerve. Square wave pulses were used with a frequency of 100 Hz and pulse width of 0.1 ms. During the period of electrical stimulation and for a short duration following, the investigators developed hypalgesia in the distribution of the nerve. With this positive foundation, they then treated eight patients with severe cutaneous pain with peripheral nerve stimulation.

Since this initial report, PNS has attracted significant interest, both as applied to specific nerves and as applied to subcutaneous peripheral nerve fields. Craniofacial pain is likely the most common indication for PNS today, targeting the occipital, supraorbital, and infraorbital nerves. However, a broad range of targets and surgical techniques have been used in an effort to treat chronic regional neuropathic pain.

The gate control theory provides a useful framework to understand a possible mechanism for PNS. The gate control theory postulates that both large- and small-diameter axons provide sensory input to the cells of the substantia gelatinosa in the dorsal horn of the spinal cord. The substantia gelatinosa serves as a gate control system that modulates the input from the large- and small-diameter fibers before they influence the first central transmission (T) cells in the dorsal horn. The summation of the excitatory input from the large-diameter fibers and the inhibitory input from the small-diameter fibers modulates the overall inhibitory activity of the substantia gelatinosa. Hence, an increase in the excitatory input from the large-diameter fibers will, in turn, increase the inhibitory output from the substantia gelatinosa, which will decrease the nociceptive output from the T cells to higher processing centers.

Although the gate control theory can provide one basis for pain relief seen with stimulation of large-diameter afferent fibers, other experiments have shown that the gate control theory does not account for all of the interactions between the large- and small-diameter afferents.³

Other mechanisms for PNS have also been proposed. Campbell and Taub⁴ performed transcutaneous electrical stimulation of the digital nerves and recorded compound action potentials (CAP) from the median nerve. With lower amplitude continuous stimulation (10–12 V, 100 Hz, 1 ms pulse width), the authors found an increased threshold for touch, but not for pain. With the amplitude of stimulation increased to 50 V, but with periodic stimulation, patients experienced pain, and the CAP demonstrated the presence of Ad waves. When the stimulus was changed from

periodic to continuous, the sensation of pain disappeared along with the Ad waves seen on the CAP. This experiment suggests that a peripheral mechanism may also account for the mechanism of PNS.

More recently, Ristić et al⁵ applied PNS to the superficial radial nerve trunk and recorded cortical laser-evoked potentials after painful stimulation. Mechanical and thermal perception thresholds were also measured. The authors found a significant reduction in the mechanical perception threshold induced by PNS due to a collision of orthodromic and antidromic Ab fiber activity. These data support the concept that a peripheral mechanism may also be involved in the analgesia attained through PNS.

■ Indications

Appropriate candidates for PNS are patients with chronic neuropathic pain in the distribution of a peripheral nerve. Electrodes are generally placed proximally to the injury site. Historically most commonly treated nerves are the ulnar, median, radial, posterior tibial, and common peroneal nerves. The last 5 years have seen a significant rise in the use of peripheral nerve stimulation for the treatment of craniofacial pain. Leads targeting the supraorbital, infraorbital, and occipital nerves have been effectively used in the treatment of these neuralgias.

Factors in evaluating a patient for PNS are:

- A demonstrated pathology for the pain complaint
- Failure of more conservative therapies, including surgery
- No significant drug dependence issues
- Adequate patient motivation and intelligence
- Clear understanding that PNS can reduce pain but cannot cure the underlying disease or problem
- Successful trial stimulation
- Pain arising from an identifiable nerve, with temporary pain relief resulting from use of selective nerve blocking techniques

Prior to considering PNS, the patient should have undergone a trial of medications for neuropathic pain and either have had no pain relief or had significant side effects from the medications, which precluded their use. As with all implantable neuromodulatory devices for pain, implantation of a PNS is predicated on a successful trial. Depending on the target nerve, a percutaneous or open surgical approach may be used for the trial.

■ Techniques

The first step in the treatment process is performing a trial of the peripheral nerve stimulation. Both percutaneous and open surgical techniques can be used for placement of the trial electrodes. A criterion of 50% reduction in pain is accepted as a successful trial.

The technique for the placement of leads for the occipital, supraorbital, and infraorbital nerves is detailed elsewhere. These leads are most commonly placed using a percutaneous technique. A percutaneous technique has also been applied for PNS trials targeting the median, radial, ulnar, peroneal, and posterior tibial nerves. For percutaneous trials targeting larger nerves of the upper and lower extremities, the use of ultrasound guidance can be beneficial to ensure proper lead location.⁶ A percutaneous technique can be performed under monitored anesthesia care or under general anesthesia. Radiographic placement for most percutaneous trials is usually adequate to ensure appropriate location, obviating the need for an awake patient and hence maximizing patient comfort.

An open surgical technique is the most commonly used approach in PNS for targeting nerves of the upper and lower extremities. Microsurgical technique is used to expose a segment of the involved nerve proximal to the injury and site of pain. Generally, a span of 4 to 5 cm of the nerve must be dissected free from the surrounding tissues to allow room for the trial electrode. Some authors have advocated the use of a fascial sling to serve as a barrier between the electrode and the nerve, analogous to the role the dura serves in spinal cord stimulation. However, with the ability to more finely control the amplitude of stimulation with the newer pulse generators, this may not be necessary. If a fascial sling is desired, a piece of fascia 4 cm × 1 cm may be harvested locally. The electrode can then be placed on the nerve and secured to the surrounding fascia to minimize the risk of lead migration. **Fig. 5.1** illustrates the technique for implantation of a paddle electrode for ulnar nerve stimulation. Typically, a 4-contact electrode is used for peripheral

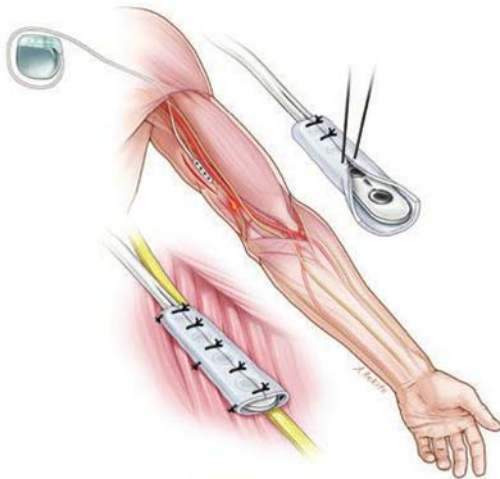


Fig. 5.1 Surgical technique for implantation of a paddle electrode for ulnar nerve stimulation. After exposure of an adequate segment of the ulnar nerve, a fascial cuff is harvested and secured around the electrode. The electrode may then be placed above or below the ulnar nerve and tacked down to the surrounding fascia to prevent migration.

nerve stimulation, but as technology continues to evolve, 8- or 16-contact paddles can also be considered.

If a percutaneous technique is chosen, the lead used for the trial will be anchored to the exit site. Most device manufacturers offer a locking anchor for percutaneous leads, which can prevent migration during the trial. After a trial of 5 to 7 days, the percutaneous lead can be removed in clinic. If the trial was successful, the patient will return to the operating room for placement of the permanent lead and implantable pulse generator (IPG). If an open surgical technique is chosen for placement of the trial electrode, an extension cable is connected to the surgically placed paddle lead, and the extension cable is externalized. This maintains the sterility of the paddle electrode. If this trial is not successful, the patient is returned to the operating room for removal of the extension cable and paddle electrode. If, however, there is a successful trial, the patient is returned to the operating room, the externalized extension cable is removed, and the paddle electrode is then connected to an IPG.

Various locations can be used for the IPG, the most common being the infraclavicular, subcostal, and posterior superior buttock regions. If the targeted nerve is in the lower extremity, the IPG may also be placed in the lateral thigh. Depending on the length of the lead used (commonly between 40–70 cm), an extension cable may be necessary to connect the IPG.

PNS typically uses a lower voltage than spinal cord stimulation, with therapeutic levels of stimulation generally between 0.5 and 3 V. Common pulse widths are between 120 and 400 μ s and rates range from 50 to 80 Hz. Intermittent cyclical stimulation is often adequate and can help to prolong battery life and, in the case of a rechargeable IPG, increase the interval between rechargings.

■ Outcomes

Peripheral Nerve Stimulation

Hassenbusch et al.⁷ prospectively treated 32 patients with PNS for stage III reflex sympathetic dystrophy. Preoperative symptoms included allodynia, deep burning pain, vasomotor tone changes, trophic changes, motor weakness, temperature changes, temporary improvement after sympathetic blockade, and a history of trauma to the affected nerve. Targeted nerves included the median, ulnar, radial, common peroneal, and posterior tibial. Of the 32 patients who underwent a trial, 30 (94%) underwent permanent implantation. Follow-up ranged from 2 to 4 years, and long-term good or fair pain relief was seen in 19 patients (63%).

In 2000 Novak and Mackinnon⁸ reported their experience with PNS in patients who had suffered an injury to a peripheral nerve. Seventeen patients were treated; 12 underwent PNS of the upper extremity and 5 of the lower extremity. Targeted nerves included the ulnar ($n = 10$), median ($n = 1$), radial ($n = 1$), and posterior tibial ($n = 5$). Over a mean follow-up time of 21 months, excellent pain relief was reported by 5 patients, good by 6 patients, fair by 4 patients, and poor by 2 patients. Of the 12 patients not working preoperatively, 6 returned to work.

In 2007 Mobbs et al.⁹ reported their experience with 45 patients who underwent a trial of peripheral nerve stimulation in the treatment of pain due to nerve trauma, iatrogenic injury from surgery or percutaneous interventions, or

continued pain after peripheral nerve tumor resection or decompression. Only 4 patients failed the trial and did not proceed to permanent implantation. Nerves targeted included the median ($n = 11$), ulnar ($n = 10$), brachial plexus ($n = 9$), radial ($n = 3$), suprascapular ($n = 1$), common peroneal ($n = 2$), sural ($n = 2$), posterior tibial ($n = 2$), and sciatic ($n = 1$). Over a mean follow-up of 31 months, an overall good result was seen in 23 out of 38 patients (61%). The mean verbal pain score (VPS) preoperatively was 9 and, on 1-month follow-up, was 5.1. This level of efficacy was maintained with a mean VPS of 5.2 at last follow-up.

Anatomically guided percutaneous and ultrasound-guided percutaneous techniques have also been successfully applied for PNS. Small series of patients have been treated for ilioinguinal neuralgia^{10,11} and testicular pain.¹² In 2009 Huntoon and Burgher⁶ reported their series of eight patients who underwent ultrasound-guided percutaneous trial of PNS for neuropathic pain that responded to a peripheral nerve block. Targeted nerves included radial ($n = 3$), ulnar ($n = 2$), posterior tibial ($n = 1$), median ($n = 1$), and common peroneal ($n = 2$). Six of eight patients had a successful trial and underwent permanent percutaneous implantation. Over a minimum follow-up of 8 months, 83% of the patients had greater than 50% pain relief.

Subcutaneous Field Stimulation

Subcutaneous field stimulation (SFS) is a relatively new procedure, in practice for over a decade now. It involves implanting leads in the painful area itself rather than directly over a specified peripheral nerve (**Fig. 5.2**). Recently, the use of two parallel electrodes placed on either side of the painful region, one serving as anode and the other as cathode, has been described¹³ (**Fig. 5.3**). The procedure for the implantation of the leads and IPG are similar as for percutaneous PNS,



Fig. 5.2 Subcutaneous field stimulation (SFS) applied to a patient with localized pain following thoracic ganglionectomy. Leads are placed in the region of maximal pain intensity.

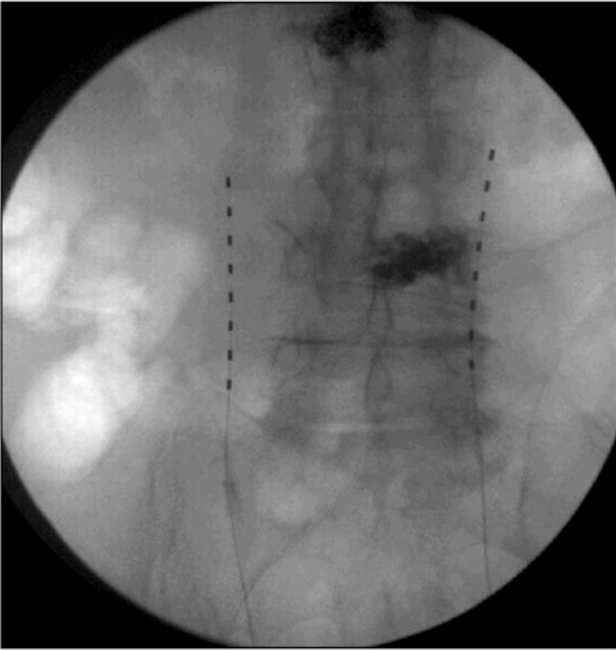


Fig. 5.3 Subcutaneous field stimulation (SFS) applied for axial lower back pain that did not respond to spinal cord stimulation (SCS). Two parallel electrodes are placed, each centered on the point of maximal pain, marked preoperatively.

detailed above. Patients with axial low back pain, neck pain, or intercostal neuralgia may be good candidates for SFS.

In a large, multicenter, retrospective series of 119 patients from seven Austrian centers, Sator-Katzenschlager et al.¹⁴ reported outcomes for patients treated with low back pain ($n = 29$), failed back surgery syndrome ($n = 37$), cervical neck pain ($n = 15$), postherpetic neuralgia ($n = 12$), and other focal pain syndromes. Of the 119 patients in the trial, 111 patients experienced more than 50% reduction in pain on the numerical rating scale (NRS) in the 1- to 2-week trial period. After permanent electrode implantation 92% of patients showed continued pain relief 3 months after the procedure, with mean NRS reduction from 8.2 to 4.0. The authors also noted a reduction in the strength and dosages of opioids used. Of the patients implanted, 24% developed complications, including infection in 7 patients (6%), lead migration in 14 patients (13%), and lead fracture in 6 patients (5%).

SFS is also being increasingly used as an adjunct to spinal cord stimulation (SCS) that fails to relieve axial symptoms adequately. Hamm-Faber et al.¹⁵ treated 11 patients with failed back surgery syndrome in whom SCS was not adequate for treating low back pain. In 9 of these patients SFS was used along with SCS to treat the pain, and in 2 patients SFS was used alone. Significant improvements were

seen in pain scales and in the usage of pain medication, adding support for the use of SFS in patients with localized pain syndromes.

■ Conclusion

Peripheral nerve neurostimulation has evolved over the last 30 years to become an important therapy in the control of intractable pain caused by peripheral mononeuropathies and sympathetically mediated pain syndromes. The growing successful experience with PNS for craniofacial pain suggests a wide variety of applications for localized pain control of conditions including postthoracotomy pain, postherniorrhaphy pain, and incisional neuroma pain. Newer percutaneous techniques using multipolar wire electrodes placed adjacent to peripheral nerves without the need for extensive dissection should help to foster PNS as a reasonable neuroaugmentation alternative to more destructive methods of chronic pain control.

Editor's Comments

Peripheral nerve stimulation (PNS) was the first clinical application of the gate control theory proposed by Melzack and Wall in 1965. As described in this chapter, it has been used ever since to treat neuropathic pain of suspected peripheral origin.

Although the initial indications for PNS were directed at discrete nerve injuries, over time the method has been generalized to treat painful regions. The bridge to that application was occipital nerve stimulation (ONS) for occipital neuralgia because these stimulation electrodes were placed simply in the *vicinity* of the greater and lesser occipital nerves, the hope being that the nerves would be recruited by stimulation prior to the overlying musculature. The use of this approach for supraorbital and infraorbital neuralgias is similar.

As described by the authors, PNS is now being applied subcutaneously to painful regions, such as the low back, with no attempt to target a specific nerve. Subcutaneous field stimulation (SFS) is being used to treat axial back and neck pain. I doubt this is what Sweet and Wall had in mind when they stimulated their own infraorbital nerves.

The quality of evidence to support these procedures is still rather low. Follow-up is short even for retrospective trials, sometimes measured in months, and there have been no prospective randomized trials. Further, all the reports have been from the implanting surgeons. It would seem a simple matter to conduct a high-quality trial of PNS, which could incorporate no stimulation, or off-target stimulation as a control. Unbiased follow-up by a third, unconcerned party would add to the validity, as would the use of accepted outcome measures. The fact that this has not been accomplished in the almost 50 years since the inception of this technique attests to the facts that patients who are considered for PNS are uncommon in most centers, and that the potential health impact of PNS is so marginal as to be below the radar of most granting agencies. It is difficult for any center to mount a reasonable trial under these circumstances.

Unless PNS is subjected to better analysis, it is likely that insurance or governmental funding for this procedure will continue to diminish, and that it will not remain part of our surgical armamentarium. At least in the United States, the impact of health care reform will focus attention on these unproven and expensive modalities. It may be time for the manufacturers of these devices to support a prospective trial, coordinated by a contract research organization (CRO), to determine if these procedures can stand up to the scrutiny of a well-conducted study. A study like this would be viewed as a positive, and long-overdue, development in our field, regardless of the outcome.

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6

Occipital Nerve Stimulation

Richard L. Weiner

Neurostimulation for chronic headaches has evolved significantly over the years since the author's first intraoperative observation in 1993¹ that subcutaneous stimulation via percutaneously placed "off-the-shelf" spinal cord stimulator wire electrodes produces paresthesias in a dermatomal or myotomal distribution without the need for direct peripheral nerve contact. Additionally, these paresthesias were capable of reducing pain perception in a manner similar to spinal cord or direct peripheral nerve stimulation with generally lower voltage and similar frequency and pulse width settings.

The early application of this discovery was in treating a group of patients presenting with intractable occipital neuralgia and reported in a seminal paper in 1999.² The diagnosis of occipital neuralgia is felt by headache neurologists to be a relatively rare occurrence in a typical headache practice. It is described as a paroxysmal, sharp, lancinating head pain in the distribution of a greater, lesser, or third occipital nerve very similar to a trigeminal neuralgia attack. This classification was certainly not well understood in the neurosurgical literature at a time when most neurosurgeons would only encounter these patients as referrals for occipital nerve excision due to intractable pain. The original group of patients reported in the 1999 publication were initially diagnosed with occipital neuralgia from a variety of etiologies, including head injury and cervical trauma. The publication of a new headache classification,³ timely input from neurology colleagues, and a repeat evaluation of the original reported occipital nerve stimulation (ONS) series⁴ clarified that most patients presenting with occipital headaches in the study group were actually diagnosed as having chronic migraine headaches as a primary headache condition rather than secondary headaches, under which occipital and other cranial neuralgias are listed. Still, the initial excellent outcomes from these intractable headache patients, coupled with subsequent reports from numerous clinical investigators,⁵⁻⁷ has led to a resurgence of activity in the field of chronic pain management, with the recognition that the subcutaneous tissues throughout the craniofacial, body, and extremity regions are viable areas in which to consider the use of neurostimulation for treatment of a large number of chronic pain conditions.

Whereas neuromodulation may be indicated for a variety of craniofacial syndromes, including cluster headaches, posttraumatic neuralgia, trigeminal neuralgia, and persistent idiopathic facial pain,⁸⁻¹⁰ the scope of this discussion, although limited to pain in and around the occipital region, neurostimulation techniques, outcomes, and future trends, is applicable to a much wider array of indications.

■ Indications

ONS delivers a small electrical charge, producing usually agreeable paresthesias when placed subcutaneously in the region of one or more occipital nerves,

without the need for direct nerve contact to control pain in patients unresponsive to medications or other conservative treatment. The typical device is a spinal cord stimulator system comprising an implantable pulse generator (IPG; chest, abdomen, or upper buttock region placement) attached to extension leads tunneled and connected to subcutaneously placed, standard multicontact electrodes directed either horizontally or diagonally from the skull base at approximately the C1 level to cover the areas of maximal pain in the occipital regions as identified by the patient. Continuous or intermittent stimulation has been used for pain control.

Subcutaneous placement of one or more multicontact leads, using either a wire or a paddle¹¹ configuration, is usually surgically straightforward and avoids the need for surgical cut-down to expose major peripheral nerves. This has been a major factor in the emergence of this form of neuromodulation as a safe, effective, and easily reproducible technique for pain control. Similar to evaluations for spinal cord stimulator implants, a temporary trial period of up to several weeks is usually indicated to predict success with a permanent implanted device. Patients have time to decide if the paresthesias are agreeable and produce pain relief both in sedentary and active modes. It also gives the implanter some idea of the stimulation parameters that might be required with a permanent device.

Current indications include:

- Occipital neuralgia
- Chronic daily occipitally mediated migraine headaches (transformed migraine)
- Intermittent migraine headaches with occipital triggers
- Chronic neuropathic pain—occipital distribution
- Posttraumatic neuropathic pain (blunt trauma, postoperative incisional pain)
- Cervicogenic occipital pain

Relative contraindications include:

- Debilitating chronic systemic diseases
- Excessive narcotic intake/dependency
- Psychological issues predictive of poor outcomes
- Diffuse pain in about the craniofacial region
- Active infection

■ Techniques

Trial Stimulation

Successful percutaneous wire trial electrode placement (temporary or permanent) depends on several factors:

- Subcutaneous space identification
- Electrode contact depth
- Outline of greatest painful areas
- Appropriate patient

The patient is given short-acting IV sedation either in the prone or lateral position followed by Tuohy needle or 14-gauge angiocatheter insertion (local anesthetic infiltrated at the skin entrance site) on either side of the midline (or unilaterally if indicated) inferior to the area of identified pain at the upper cervical region posteriorly. The needle/catheter is directed in the subcutaneous space cephalad from left to right and right to left of midline (**Fig. 6.1**) to just beyond the area of defined maximal pain, which is usually a couple of finger breadths off the midline for greater occipital nerve pain and more lateral for lesser occipital nerve pain. It is paramount to avoid any damage to the dermis layer when the needle or catheter is being inserted for both temporary and permanent electrode placement to avoid the risk of erosion and subsequent infection. The patient is awakened and temporary stimulation current is then applied to verify electrode location. The patient should feel paresthesias into the scalp area around the electrode array with polarity shifting utilized to maximize effective paresthesia coverage around the painful areas. A grabbing sensation or muscle twitching usually indicates the electrodes are deep to the fascia with the cervical musculature and will need to be repositioned. Leads placed too superficially within the dermis will lead to erosion and possible infection (temporary and permanent electrode placement). The leads exiting the insertion site are



Fig. 6.1 Crossed temporary leads directed superolaterally.

then sutured to the skin with supplied anchors and antibiotic dressings applied (**Fig. 6.2**). The externalized lead extensions must then be secured with enough tape and lead coiling to prevent lead pullout.

Permanent Implantation

The temporary electrodes should be removed in the clinic by the operator, who can then carefully interview the patient regarding stimulation response and pain control as well as medication usage and the desire to continue toward or decline a permanent implant. Permanent placement of electrodes is usually delayed at least 2 weeks to allow healing around the puncture sites and minimize the risk of infection. Care should be taken to avoid incisions in or immediately adjacent to the healing puncture sites. For patients with permanent temporary leads, the externalized lead extensions can be carefully cut so the distal lead connects to permanent subcutaneous extensions tunneled to the IPG.

Suitable candidates for implantation should be made to understand some caveats to the permanent occipital nerve stimulation or other subcutaneous per-



Fig. 6.2 Temporary lead placement.

manent implant device. Some patients may be referred for permanent implants after having been in a trial elsewhere, and it is imperative that they be properly counseled regarding their expectations and limitations of the device.

Limitations

As of this writing, ONS and other subcutaneous neurostimulation procedures remain off label in the United States but have received approvals in Europe and Australia.

1. No system to date is completely magnetic resonance imaging (MRI) compatible, and patients requiring periodic MRI evaluation for unrelated medical issues may be unable to comply.
2. Cautions apply with regard to operating machinery and driving, similar to those for spinal cord stimulation because there could be a rare voltage or current surge that might startle the patient.
3. The risks of infection and lead migration or extrusion with current systems remain the chief complication.
4. Stimulation tolerance with loss of pain control can occur in up to 20% of cases over time.

Expectations

1. There is a spectrum of utilization and response to ONS therapy. This includes intermittent use when a headache onset is perceived, all the way to constant use with chronic daily headaches.
2. These devices may be considered as adjunctive therapy for many intractable headache sufferers, which will still require other treatment modalities to minimize pain.
3. The implants are usually not curative.
4. Underlying behavior and narcotic intake issues still need to be treated and monitored.

Procedure Considerations

Implantable Devices

Most ONS implants are comprised of a rechargeable pulse generator, lead extension(s), and one or more multicontact wire electrodes. Paddle electrodes have also been used in some patients who may need a broader region of stimulation or who have had electrode migration issues, although some surgical dissection is necessary in the subcutaneous space for correct placement. There are currently three main power source options available for the implanter to consider: primary cell nonrechargeable IPG, rechargeable IPG, and external radiofrequency (RF) transmitter/receiver systems. Interestingly, the RF systems are on label for peripheral nerve indications. If voltage and current requirements are low during the trial, a primary IPG might be advantageous in the older patient group to reduce noncompliance from perceived complexities of periodic battery recharging.

Head Position

Although the prone position in a padded head holder is technically the most straightforward way to implant electrodes and tunnel extension wires (**Fig. 6.3**), many anesthesiologists are uncomfortable with airway control during sedation. Also, the IPG implant site would need to be posterior as well, usually in the upper buttock region, with long primary electrode or extension wires tunneled from the neck through a sometimes difficult cervicothoracic subcutaneous junction. The lead extensions may stretch for 3 inches or more with forward bending postoperatively, possibly contributing to a high lead migration rate. Either the lateral or supine position with head turned to the opposite side can be used to implant bilateral occipital electrodes tunneled to an upper chest IPG placement with much less lead movement/migration potential depending on the anchoring technique.

Surgical Prepping and Draping

To minimize the risks of infection, the implanter must take great care to remain meticulous throughout the procedure, including the initial prep and draping techniques. Hibiclens (Mölnlycke Health Care, Norcross, GA, USA) showers the evening before surgery have been shown to be beneficial. Suboccipital hair should be shaved with clippers only. The use of razors has been shown in multiple studies to increase infection rates due to bacteria entering the minute skin abrasions produced by the razor. Alcohol-based preps with Steri-drape (3M, St. Paul, MN, USA) coverage of all skin surfaces is recommended (**Fig. 6.4**).



Fig. 6.3 Prone position with midline incision and maximal painful areas mapped bilaterally.



Fig. 6.4 Antibacterial draping.

Generator Site

Generator placement influences patient positioning and the potential for post-operative electrode migration. Additionally, cosmetic appearance and IPG pocket comfort for chest and buttock placements, respectively, are important considerations. The following are typical implant locations:

1. **Upper chest.** Lateral or supine positions favor this location. Take special care when tunneling across the lateral neck region and over the clavicle to avoid possible accessory nerve, carotid artery or jugular vein injury, or inadvertent thoracic cavity puncture with subsequent pneumothorax. Women with large breasts may be a challenge in placing the IPG appropriately.
2. **Upper buttock.** This common area for placement facilitates electrode/extension tunneling to the IPG. Cautions include the need to avoid placing the IPG too low in the buttock; to avoid medial placement, which could damage the cluneal nerve; and to be aware that flexion tension on the electrode/extension appears to contribute to a relatively high incidence of lead migration.
3. **Abdomen.** The lateral position facilitates placement, probably with less risk of lead migration.
4. **Lateral thigh.** This location is useful for spinal cord stimulation but is not practical for ONS.

Electrode Tunneling and Fixation

Electrode migration has been reported in up to 24% of implants, no doubt due to the highly mobile upper cervical region, and is probably the most problematic part of the procedure. Various anchoring devices are available, which differ

depending on the electrode manufacturer and achieve variable degrees of secure fixation. Some implanters prefer to tie sutures to looped electrodes as part of lead fixation without anchors, using the surrounding tissues to “squeeze” the electrode at the suture point. Strain relief loops, both proximal at the electrode incision site and distally with looped extension wires, have been used to minimize direct tension on the electrode contact array. Using medical-grade silicone glue can be helpful within certain anchor/electrode contact points. Newer “Chinese handcuff” and bumpy-type anchors offer improvement over traditional cuff anchors. Minimizing electrode/extension lengths to the IPG pocket (i.e., chest placement or miniaturization of power sources placed closer to the electrodes) might significantly reduce the migration problem.

Intraoperative Stimulation Evaluation

The purpose of controllable short-acting sedation is to allow quick and accurate positioning of one or more electrodes into the subcutaneous tissues identified as subjacent to the maximal areas of pain/tenderness in the occipital scalp innervated by the occipital nerve system. This in turn allows direct patient feedback during stimulation testing intraoperatively to verify electrode placement in terms of depth and painful region. Patients should be queried as to whether the stimulation sensation is one of paresthesias as opposed to burning or grabbing discomfort. Feedback that the paresthesias cover the majority of their most painful areas allows for subsequent postoperative programming modes to be entered into the IPG for more complete pain coverage.

Stimulation Parameters

The following settings are a good starting point for intraoperative and postoperative programming with the cathode steering the polarity considerations:

- Lead polarity
- Voltage: 1 to 4 V
- Frequency: 30 to 60 Hz
- Pulse width: 120 to 240

It remains to be seen if ultra-high-frequency stimulation in the 10,000-Hz range will offer any advantages for long-term pain control as these devices become available for study.

Postoperative Management

ONS implantation surgery is usually an outpatient procedure; however, mitigating circumstances could include excessive postoperative pain from surgical dissection for paddle placements, and issues relating to operating on patients with long-term narcotic intake and tolerance problems. These patients might require a more extended hospital stay for pain stabilization. Many implanters will use cervical collars for several weeks to help minimize excessive neck movements postoperatively. Patient instructions, including recharging procedures, should probably be reiterated either in person or over the phone a day or two after the

procedure because many patients have difficulty handling information following same-day sedation. Dry, sterile dressing changes are important to minimize wound infection.

Implant Technique

ONS surgical implant techniques have been described in multiple journal articles, textbooks, and monographs. In place of a step-by-step procedure, the following technical observations, gained from 20 years of experience by the author, might be of assistance to the implanter. The list is by no means exhaustive but merely represents some personal reflections.

1. The subcutaneous space in the occipital region usually accepts a tunneling electrode introducer (Tuohy needle or angiocatheter) without much difficulty at the right depth. Larger patients with greater subcutaneous fat are obviously easier to implant but may have a greater risk of migration. Consider ultrasound guidance for thinner patients.
2. When advancing the introducer within the subcutaneous tissues, follow the needle tip with finger palpation to be sure there is no dermal penetration, which could set into motion underlying tissue damage resulting in electrode erosion and infection. Do not underdrape the suboccipital region because an electrode could inadvertently poke through the skin during a lateral traverse under the drapes (**Fig. 6.5**).
3. Be meticulous with electrode anchoring to minimize migration.
4. Tunneling can be dangerous. Some of the supplied tunneling tools have very sharp tips and require careful handling. I am aware of a case of severe

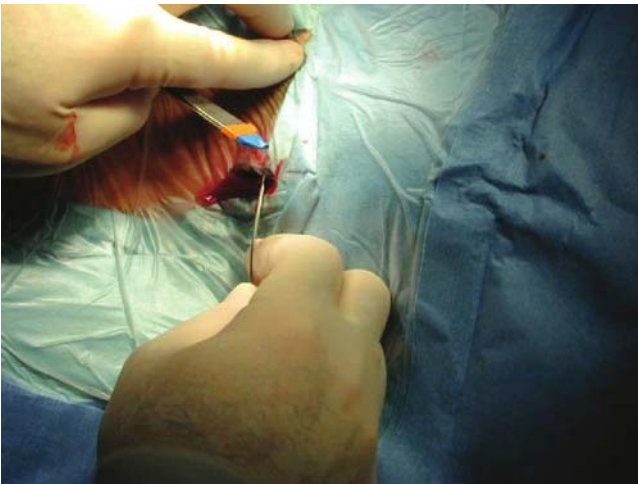


Fig. 6.5 Gentle subcutaneous needle insertion while using left index finger to monitor needle tip to avoid placing too superficially or too deep into the fascia.

brain injury from a tunneling tool entering the foramen magnum during tunneling in a cephalad direction to connect an extension cable.

5. Prone positioning requires an experienced anesthesiologist and proper sedation to avoid airway difficulties or wound contamination from undersedation and excessive patient movements.
6. Chest IPG placement, when feasible, may be preferable over the other implant sites due to migration risk.
7. Always tunnel the electrode a centimeter or two beyond the area of maximal tenderness because tugging during on-the-table stimulation is only by electrode pullback.
8. Many patients with bilateral greater occipital nerve pain can be treated with a single 8-contact electrode placed across the midline using a single lateral incision.
9. Patient sedation can be challenging, especially in the presence of narcotic tolerance. IV ketamine can be quite effective in selected cases.
10. Counsel patients that ONS therapy is adjunctive and not curative to avoid misperceptions and disappointment.

Complications

Possible complications of ONS surgical implantation include:

- Migration, erosion
- Infection, hematoma
- Equipment malfunction/breakage
- Altered, attenuated, or failed response to stimulation
- Persistent incision site pain
- Wrong patient

Surgical complications for any procedure are multifactorial; however, better electrode and power source designs incorporated into the locally placed electrode with proper implantation technique should positively address most of these potential complications.

■ Outcomes

Numerous nonblinded, single-center studies in the literature claim a long-term success rate, defined as greater than 50% pain reduction in approximately 70 to 80% of implanted patients, concluding that ONS is a safe and effective therapy. Reduction in headache intensity and frequency has also been observed in multiple series. There have been three major device manufacturer-sponsored multicenter studies^{12–14} to date that attempted to demonstrate the safety and efficacy of ONS in a migraine headache population for FDA device approval. None of these studies, however, achieved a statistically significant pain reduction endpoint of 50%. The best outcomes were from the St. Jude Genesis study¹²:

- 28% decreased headache days (7 d/mo) versus 4% (1 d/mo) placebo group
- 42% pain relief versus 17% placebo group

- 53% excellent or good pain relief versus 17% placebo group
- 67% improved quality of life versus 17% placebo group
- 51% satisfaction with headache relief versus 19% placebo group
- However, primary endpoint of 50% not met
- Difference between active and placebo groups statistically significant at 40% pain reduction level
- CE Mark achieved September 2011

It has been argued that statistical significance at 40% still represents a significant improvement for a group of chronic headache sufferers with little or no other treatment options who would benefit greatly from ONS. Unfortunately, most insurance companies will continue to avoid reimbursement for the devices and procedure due to its current off-label status. FDA approval following subsequent controlled studies is the goal of physicians, patients, and device companies.

Mechanisms of Action

Occipital nerve stimulation is actually the delivery of neuromodulatory electrical signals to subcutaneous tissues innervated by branches of greater, lesser, or third occipital nerves, either singly or in combination, unilaterally or bilaterally. When stimulation is applied, paresthesias are felt in the distribution of sensory representation of that nerve. The beneficial effects for occipitally mediated headache syndromes appear to involve the following elements:

- Subcutaneous electrical conduction
- Local innervation
- Dermatomal stimulation
- Myotomal stimulation
- Sympathetic stimulation
- Neurochemistry
- Blood flow alteration
- Trigemincervical complex

Because the occipital nerves arise from the C2 and C3 nerve roots, it has been postulated that antidromic neurostimulation of these roots via the occipital nerve branches interacts with the descending tract of the trigeminal nerve into the upper cervical region with inhibition of interneurons in the trigemincervical complex, and interrupts or decreases transmission of pain signals to the thalamic and frontal lobe recognition areas.^{15,16}

■ Conclusion

There continues to be significant interest and excitement throughout the pain management world in the utilization of ONS and other subcutaneous peripheral stimulation techniques to treat intractable pain conditions. The success of this treatment modality will require a combination of carefully designed and implemented outcome studies, clarification of appropriate implant indications, and a new generation of implant devices tailored to ONS and other peripheral sites.

Editor's Comments

Dr. Weiner is the pioneer not only of occipital nerve stimulation (ONS), but also of subcutaneous stimulation in general. In the United States ONS remains an off-label but otherwise viable treatment for occipital neuralgia. I continue to have difficulty separating this diagnosis from "cervicogenic" headache, but given that ONS is a testable surgical therapy, I would not hesitate to conduct trial stimulation in a patient who complains of suboccipital pain, whether intermittent or constant.

ONS is 20 years old, but as Dr. Weiner points out, more data from a randomized prospective series will be necessary to fully legitimize this technique and potentially gain FDA approval. The lack of such approval is clearly hampering the application of this therapy.

There has been much speculation, and enthusiasm, about the use of ONS for other headache syndromes—migraine in particular. As reviewed in this chapter, results for headache have been disappointing, and thus far no randomized prospective trial has met its primary endpoint for success.

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Trigeminal Neurostimulation for Neuropathic Facial Pain

Charles Nelson Munyon and Jonathan Miller

The face occupies only 4.5% of the body surface but is uniquely important for human identity, playing an indispensable role in basic survival functions like breathing and eating as well as complex communications involving vocalization and expression. Facial pain can interfere with all of these functions and is a source of considerable morbidity for patients affected by it. In particular, facial pain due to trigeminal nerve injury often does not respond adequately to medications and as a result can be particularly difficult to treat.¹ Neuromodulation techniques involving stimulation of the trigeminal nerve or its branches can be helpful to ameliorate certain types of neuropathic facial pain.

■ Trigeminal Neurostimulation: History and Reported Results

Stimulation of the gasserian ganglion as a means of treating trigeminal branch pain was first described in 1980 in a case series by Meyerson and Håkanson,² who published an expanded series in 1986.³ Stimulation of distal trigeminal nerve branches was first described in 2002 in a report of two patients who underwent subcutaneous stimulator placement along the course of the affected nerve for postherpetic neuralgia with significant pain relief and reduction in medication requirements 3 years after implantation.⁴ Johnson and Burchiel subsequently published a retrospective case series of 10 patients with posttraumatic trigeminal neuropathic pain ($n = 5$), postherpetic neuralgia ($n = 4$), or idiopathic trigeminal neuropathic pain ($n = 1$). At a mean follow-up of 27 months, all of the trigeminal neuropathic pain patients and 2 of the 4 postherpetic neuralgia patients reported a reduction in pain levels greater than 50%.⁵ As part of a technical note in 2005 Slavin described a series of 8 patients that was expanded in 2006 to include 9 patients treated with trigeminal branch stimulation as part of a cohort of 22 patients undergoing trigeminal, occipital, or trigeminal and occipital nerve stimulator implantation.^{6,7} More recent reports include a series of 10 patients who underwent neurostimulation for supraorbital neuralgia,⁸ a series of 3 patients with posttraumatic trigeminal neuropathic pain,⁹ and case reports of patients with posttraumatic trigeminal neuropathic pain or postherpetic neuralgia successfully managed via trigeminal peripheral nerve stimulation.^{10,11} Finally, peripheral nerve field stimulation in the mandibular distribution has been described, with relief of pain at 1-year follow-up.¹² All of these reports indicate pain relief greater than 50% at last follow-up, which varied from 6 months to 3 years. There were no major complications reported in any of the reports, although sev-

eral patients required revision or removal of their stimulator systems because of lead migration, skin erosion, or infection.

■ Indications

Selection of an appropriate treatment for facial pain requires accurate determination of the diagnosis. Since neurostimulation as a rule is more effective for neuropathic than for nociceptive pain syndromes, consideration should be given to possible sources of nonneuropathic pain, particularly in the distributions of the maxillary and mandibular nerves, where causes may include odontogenic pain and temporomandibular joint disease.¹³ Once facial pain is confirmed to be neuropathic in etiology, it is often helpful to identify the precise subtype according to the framework first proposed by Burchiel in 2003 and expanded upon by Eller, Raslan, and Burchiel in 2005.^{14,15} The patient's symptomatology and relevant medical history are used to differentiate two broad categories of pain: (1) trigeminal neuralgia pain due to idiopathic trigeminal neuralgia (types 1 and 2) or resulting from a central demyelinating process such as multiple sclerosis (symptomatic trigeminal neuralgia), and (2) neuropathic pain due to nerve or ganglion injury from nerve trauma (trigeminal neuropathic pain), iatrogenic lesioning (trigeminal deafferentation pain), or herpes zoster (postherpetic neuralgia). Trigeminal stimulation is generally performed for the second category. Another important consideration is preservation of sensation because facial pain associated with significant sensory loss is unlikely to improve with trigeminal nerve stimulation, although some hypesthesia is not a contraindication.

Prior to consideration of any neuromodulation procedure, all potentially remediable causes of pain need to be investigated and excluded. Even if an appropriate workup has been completed, it remains essential that all relevant data be reviewed to ensure that nothing has been missed. Depending on the distribution and character of pain, referral to a neurologist, otorhinolaryngologist, ophthalmologist, or oral and maxillofacial surgeon may be indicated. The patient needs to have had an adequate trial of nonsurgical treatment, which may include a combination of opiates, anti-inflammatories, anticonvulsants, neuroleptics, or antidepressants as well as injection of local anesthetic or botulinum toxin. Therapies such as transcutaneous electrical stimulation, meditation, or psychological counseling may also play a useful adjunct role. All patients should be screened for psychological contraindications such as substance dependence, unrealistic expectations, or presence or pursuit of secondary gain. Medical contraindications include bleeding diathesis, immunodeficiency, and any condition for which the patient will likely require future magnetic resonance imaging of the head or neck.

The indications for consideration of trigeminal branch stimulation are otherwise similar to those for peripheral nerve stimulation elsewhere in the body: (1) pain should localize to the distribution innervated by the nerve(s) in question, and (2) the nerve must be in continuity proximal to the planned site of stimulation, with at least partial preservation of sensory function, although some hypesthesia may be present. Some authors advocate a trial of pharmacologic blockade or transcutaneous electrical stimulation prior to trial implantation, but the predictive value of these tests has not been definitively established.

■ Techniques

Supraorbital and Infraorbital Nerve Stimulation

Prior to implantation of a permanent stimulator, a trial period of stimulation should be undertaken to assess whether the patient derives adequate pain relief to justify implantation of the permanent system. The trial and permanent implantation are outpatient procedures and may be performed in either of two ways: (1) placement of a temporary externalized percutaneously located lead followed by subsequent implantation of an entirely new system, or (2) placement of an anchored lead with a temporary externalized extension that is discarded in a second operation when the generator is placed. The main advantage to the second approach is that the trial lead is also used for the permanent implantation, although the trial is considerably more invasive; removal in the case of an unsuccessful trial requires an additional operation, and it is necessary to use an extension cable with a bulky connector rather than connecting the lead directly to the generator. Therefore, we prefer to use the percutaneous trial method whenever possible.

Percutaneous trial placement is generally performed under local anesthesia with mild sedation to allow for patient feedback and ensure optimal coverage of the region of pain. Intraoperative fluoroscopy is helpful to confirm proper electrode placement. Once the patient is adequately sedated, the head is turned toward the contralateral side, in the case of unilateral implantation, or placed on a padded horseshoe head holder in the neutral position for bilateral implantation. The entry point is located above and lateral to the eyebrow for supraorbital nerve stimulation and just above the malar eminence for infraorbital nerve stimulation (**Fig. 7.1**). A stab wound is produced in the appropriate location. A Tuohy needle is prepared with a gentle convex curve away from the needle opening, and the needle is introduced with the stylet, with the convex curve outward to follow the contour of the skin. The skin surface overlying the needle should be carefully palpated to ensure that the needle is at the correct depth (**Fig. 7.2**). If it is placed too deep, stimulation will produce uncomfortable cramping or pulling due to muscle activation, and a course too shallow will produce unpleasant stinging instead of paresthesias. C-arm fluoroscopy is used throughout this process to verify elec-



Fig. 7.1 Ideal subcutaneous location of supraorbital and infraorbital leads. Note the entry point lateral to the orbit and that each electrode spans the respective foramen.

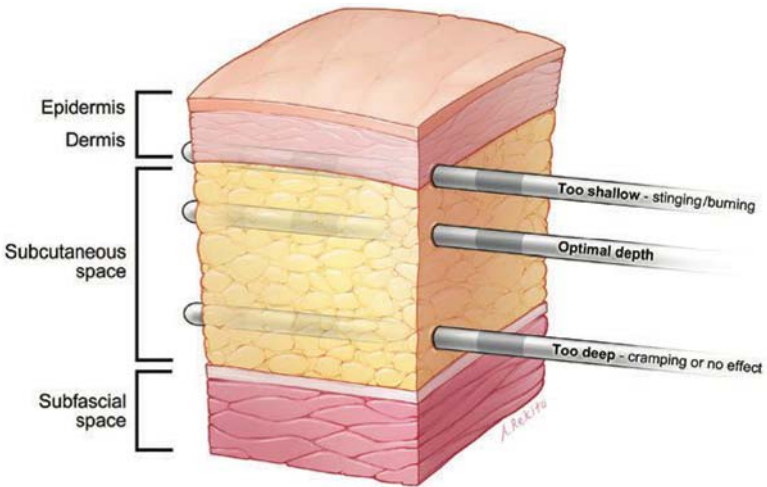


Fig. 7.2 Ideal depth of leads. The optimal depth is just deep to the dermis where paresthesias are similar to what is experienced dermatomally with epidural stimulation. A lead that is too shallow or too deep would produce different sensations and would require repositioning.

trode position. Once the needle has been advanced past the target nerve foramen, the stylet is withdrawn from the needle and the stimulation electrode is threaded into place through the needle (**Fig. 7.3**). Spinal cord stimulation electrodes with different numbers of contacts are available, but four contacts are adequate, as found on the Pisces Quad electrode from Medtronic, Inc. (Minneapolis, MN, USA). The needle is carefully withdrawn, leaving the lead in place, and the lead is connected to a stimulator to verify appropriate coverage of the area of pain without discomfort. If the electrodes are in the correct place, the patient should feel a pleasant tingling sensation that radiates along the course of the nerve. The electrode can be tunneled to an exit site above the ear by passing the stylet through the initial stab wound subcutaneously to a distal site from which the needle is passed backward around the stylet toward the initial stab wound.



Fig. 7.3 Anterior-posterior radiograph demonstrating trial placement of an 8-contact lead for supraorbital stimulation.

Removal of the stylet allows passage of the lead through the subcutaneous space to the site of exit. This process can be repeated to move the exit site further away. The externalized lead is secured to the skin at multiple locations and attached to the temporary stimulator for 3 to 10 days of trial stimulation as an outpatient procedure, after which the electrode is removed in the office.

If adequate relief is obtained during the trial, the permanent lead is placed in a separate operation (**Fig. 7.4**). The lead is placed using the same technique used during trial implantation. A 2-cm retroauricular incision is then performed with the development of a subcutaneous pocket where the lead is securely anchored prior to tunneling to the site of subcutaneous implantation of the pulse generator. The generator is generally placed in an infraclavicular location, although other sites (e.g., abdomen) may be used if necessary for cosmetic reasons or for very thin patients. A subcutaneous pocket is developed, and the generator is attached to the lead and implanted in the pocket. Prior to closure of the pocket, the generator is interrogated to ensure that the system has been properly connected. The incisions are then closed, and the patient is allowed to awaken from anesthesia.



Fig. 7.4 Anterior-posterior radiograph demonstrating placement of supraorbital and infraorbital leads. There is a lead in the foramen ovale as well for stimulation of the trigeminal nerve at that location.

Gasserian Ganglion Stimulation

Implantation of a stimulating electrode directly into the gasserian ganglion may be used to produce diffuse stimulation of the entire trigeminal nerve distribution, which can be useful to treat neuropathic pain in distributions other than the supraorbital/infraorbital, such as the orbit or V3 distribution. A smaller electrode such as a Medtronic 3389 deep brain stimulation electrode may be used for this purpose. To place the electrode, a Tuohy needle is entered into the foramen ovale via the same technique used for percutaneous rhizotomy for trigeminal neuralgia. The patient is placed supine on the operating table with the head turned slightly to the contralateral side, and rapid-acting sedation is administered. The hemiface is prepped with iodine or chlorhexidine, and local anesthetic is instilled into a point 2 cm lateral to the corner of the mouth and 1 cm inferior to the occlusal line, where a stab wound is made. A Tuohy needle is advanced into the tissue of the cheek while the needle tip is guided toward the foramen ovale using one's finger inside the patient's mouth to prevent penetration into the oral cavity. Fluoroscopy using a modified submental vertex projection can be helpful, and lateral fluoroscopy is used to verify depth of insertion. At this stage, the stylet is withdrawn and the electrode is passed into the ganglion. Test stimulation, lead externalization, and permanent placement are performed as with the supraorbital and infraorbital

leads. Rates of migration with subsequent loss of benefit are high with this approach, so careful anchoring is very important.

■ Conclusion

Intractable neuropathic facial pain can be challenging to treat due to the breadth of disorders that can cause orofacial pain and the relative resistance of many of these disorders to conventional pain management strategies. Evidence-based evaluation of these therapies has been hampered by the lack of a common classification system and marked variability in outcome measurement and length of follow-up. Standardized study is needed to better characterize the relative efficacy and cost-effectiveness of treatment. However, for carefully selected patients with localized neuropathic facial pain and preserved sensation, electrical stimulation of the trigeminal nerve or along its nerve branches can be a valuable tool to provide long-lasting relief of symptoms.

Editor's Comments

Dr. Miller has written an overview of the techniques of trigeminal branch stimulation. Although direct trigeminal ganglion stimulation has been used in the past, it is rarely employed now due to the frequent complication of lead migration. Peripheral branch stimulation, particularly supraorbital and infraorbital, allows access to trigeminal distributions without the attendant high rate of lead movement or withdrawal.

We have yet to see a randomized prospective trial of implanted trigeminal leads, although a recent double-blinded, randomized, sham-controlled prospective trial of percutaneous supraorbital stimulation showed significant benefit of stimulation compared with sham for the number of 50% responders ($p = 0.023$), number of monthly headache days ($p = 0.041$), and monthly acute antimigraine drug intake ($p = 0.007$).¹⁶ Whereas the therapeutic benefit of stimulation in this trial was modest (26%), it compared favorably with other nondrug and preventive drug therapies.

Trigeminal neuropathic pain is the clear indication for trigeminal branch stimulation. A prospective trial comparable to the one cited above for migraine would be a major contribution to the facial pain literature.

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Section II.C

Intrathecal Therapy

8

An Overview of the Rational Use of Intrathecal Analgesic Therapies

Elliot S. Krames and Tammy Penhollow

The need for the infusion of intrathecal (IT) agents should be determined by the diagnosis of the patient; the relevant patient history, including tried and failed medication management of the problem; physical examination; and imaging of the patient. It should be guided by an understanding of the appropriate indications^{1,2} for placement of IT systems, the relevant spinal anatomy, and the technique. This chapter discusses the relevant anatomy of the epidural and IT space; indications for IT therapies; the rational use of the IT delivery of baclofen for central spasticity syndromes; and analgesic medications for pain control, including the FDA-approved analgesics morphine and ziconotide (Prialt; Jazz Pharmaceuticals plc, Dublin, Ireland) and other, non-approved analgesic medications, such as hydromorphone, methadone, fentanyl and sufentanil, the local anesthetic bupivacaine, and the α_2 -agonist clonidine.

■ Relevant Anatomy

The spinal canal, containing the epidural space, the IT space, the dura, the spinal meninges, the spinal cord, fat, arteries, veins, lymphatics, nerve rootlets, nerves, and the dorsal root ganglion, is protected dorsally by the bony lamina and ligamentum flavum; ventrally by the posterior longitudinal ligament, the intervertebral disk, and the vertebral body; and laterally by the pedicles. Anatomy relevant to the placement of IT catheters for the IT delivery of opioids is that the spinal cord extends from C1–2 to L2, that the cauda equina extends from L1–2 to the sacrum, and that the dura envelops the exiting nerve roots to the dorsal root ganglia (DRG).

Cerebrospinal Fluid Dynamics and IT Catheter Fluid Flow Dynamics

Also relevant to the IT delivery of opioids is the cerebrospinal fluid (CSF) surrounding the brain and spinal cord within the subarachnoid space and produced by the ependymal cells of the choroid plexus of the brain. CSF is found in all intracerebral ventricles, spinal and brain subarachnoid spaces such as cisterns and sulci, and the central canal of the spinal cord. The rate of CSF formation in humans is about 0.3 to 0.4 mL/minute (about 500 mL/d). Total CSF volume is 90 to 150 mL in adults and 10 to 60 mL in neonates. It originates from the choroid plexus, parenchyma of the brain and the spinal cord, and ependymal lining of the ventricles.³

Agents delivered and diffused into and through the CSF are governed by the CSF circulation, CSF dynamics, cardiac output, the type of delivery of the agent into the IT space (bolus, continuous delivery, speed of delivery, where delivered, etc.),

and the physical chemical properties of the agent infused (weight, hydrophilicity, etc.). It is known that CSF returns to the vascular system by entering the dural venous sinuses via the arachnoid granulations (or villi). However, it has been suggested that CSF flow along the cranial nerves and the spinal nerve roots and through the cribriform plate also seems to be important, especially in neonates,^{4,5} since the villi in neonates is underdeveloped.

Flow of CSF is as follows: the portion of the fluid formed in the lateral ventricles escapes by the foramen of Monro into the third ventricle and then via the aqueduct into the fourth ventricle. A little CSF occurs in the central canal of the spinal cord and may be added to the intraventricular supply. From the fourth ventricle the fluid pours into the subarachnoid spaces through the medial foramen of Magendie and the two lateral foramina of Luschka. There is no functional communication between the cerebral ventricles and the subarachnoid spaces in any region except from the fourth ventricle.⁶

Two components can be distinguished in CSF circulation: (1) bulk flow (circulation) and (2) pulsatile flow (back-and-forth motion). In bulk flow theory, CSF is produced by choroid plexus and absorbed by arachnoid granulations. The force, which provides CSF movement from the ventricular system to arachnoid granulation and CSF absorption, is caused by a hydrostatic pressure gradient between the site of its formation (slightly high pressure) and its site of absorption (slightly low pressure). In pulsatile flow theory, movement of the CSF is pulsatile and results from pulsations related to cardiac cycle of the choroid plexus and the subarachnoid portion of the cerebral arteries.⁷ Because very little CSF truly circulates through the subarachnoid space, pulsatile flow, rather than bulk flow, can be measured and demonstrated by phase contrast MRI (magnetic resonance imaging).⁸

It is important to know that CSF movement in the subarachnoid space of the spinal canal changes with position. The change from the supine to the prone position increases the movement of CSF in the dorsal subarachnoid space and decreases the movement in the ventral subarachnoid space; these findings may be attributed to change in the volume of CSF in the subarachnoid space after the positional shifts of the spinal cord.⁹

Prior knowledge of IT drug distribution was based on the literature regarding spinal anesthesia and factors that presumably increase drug spread, including morphological and technical factors.¹⁰ However, because of the difference in the order of magnitude between the volume and flow rate (1 mL/30 s and 40 mL/d, respectively), most spinal anesthesia data are not applicable to chronic long-term IT drug administration. It was previously assumed that, upon its administration in the CSF, the drug would be transported by the flow of CSF and diffuse throughout the subarachnoid space to reach specific receptors in the spinal cord or the brain.

Biochemical, radiological, and experimental data refute the existence of a net CSF flow. In fact, the concentration gradient for normal small or large CSF constituent molecules is markedly rostrocaudal.^{11,12} Early radiological findings from gated MRI have demonstrated that CSF moves in a to-and-fro oscillation¹³ that is more marked in the cervical than in the lumbar region, but have not indicated the existence of a CSF bulk flow.^{14,15}

CSF velocity and amplitude varies based on which part of the spinal cord is considered.¹⁶ A pig model shows direct evidence that CSF does not circulate.¹⁷

Germane to IT catheter implantation, CSF drug distribution and spinal cord uptake are limited to the tip of the catheter and several centimeters distal to it; it does not wrap around the spinal cord, as evidenced by results from continuous IT low-flow-rate injections.^{18–21} CSF pulsations are generated by the cardiac cycle, and this is responsible for IT drug diffusion from the fluid dynamic standpoint. These oscillations produce two diffusion-enhancing phenomena: (1) steady streaming, which is related to the perturbations created by obstacles such as ligaments and nerve roots present in an oscillatory flow; and (2) enhanced diffusion, which is caused by shear forces at the liquid–solid interface and is enhanced by geometric changes, whether cross-sectional or due to an intrinsic object such as a catheter.²² Anatomical, functional, and fluid dynamic factors interact in a complex way whereby drug movements across and within nonhomogeneous spaces (IT and epidural) are difficult to predict and require different pharmacokinetic models.^{17,23} In a study of laboring women receiving IT fentanyl, analgesia duration correlated not with CSF concentration but rather with blood pressure, emphasizing the link between the cardiovascular system, CSF oscillation, and drug spread.²⁴

The CSF renewal throughout the day also has an impact, albeit unknown, on the pharmacokinetics of intrathecal drug delivery and dynamics. The clinical implications of wide variability in response to IT injections may account for a substantial portion of the discrepancies observed between single injections and continuous infusions.²⁵ A common assumption is that, for a given daily dose, the therapeutic effect is diminished with lower flow rate of higher drug concentrations. This intuitive belief is based on a number of anecdotal and mostly unpublished observations that are contradicted by two recently published randomized, controlled studies. In patients with complex regional pain syndrome (CRPS)-related dystonia, van der Plas et al.²⁶ showed that, when the daily dose of baclofen was maintained, a fourfold increase in flow rate had no effect on dystonia or pain, but adverse event (AE) rates increased. Chronic pain patients given a fourfold increased flow rate in constant daily dose also showed negative effect: It did not result in improved pain scores but was associated with a significant decrease in EQ-5D quality-of-life scores.²⁷

In summary, suffice it to say that the old and conventional wisdom that CSF moves from the spinal IT space to the ventricles by bulk flow is not supported by recent studies on the topic. CSF oscillations produced by the cardiac cycle are responsible for CSF fluid movement and for spread of IT delivered agents. Previous notions that agents spread in the CSF up and down are not supported by present knowledge, and the reality is that spread of slow delivery of agents is probably no more than at the tip and three levels above and below the tip. IT drug delivery is more complex than previously thought, and delivery and spread are governed by mode of delivery (bolus vs. continuous, slow vs. fast), the physical-chemical properties of the agent delivered (hydrophilic vs. lipophilic), cardiac output/heart rate, intrathecal obstructions such as membranes and tumors, position of the patient at any point in time, and position of the catheter within the IT space. See **Table 8.1** for the physicochemical properties of frequently delivered IT agents.

Table 8.1 Partition coefficients: the higher the coefficient, the greater the lipophilicity, and the lower the coefficient, the greater the hydrophilicity

Drug	Partition coefficient	References
Morphine sulphate	(Octanol/water), 1.42 @ pH 7.4	Infumorph 200-package insert, Baxter International
Hydromorphone HCL	(Octanol/water) 1.23	<i>Neural Blockade in Clinical Anesthesia and Management of Pain</i> , Issue 494 by Michael Cousins, Phillip O. Bridenbaugh, Lippincott Williams and Wilkins 1998
Clonidine	7.1	Hayek et al. <i>Seminars in Pain Medicine</i> 2003;1(4):238–253
Ziconotide	XLogP-10.3	Ragawski et al. <i>Neurotherapeutics</i> 2009;6(2):344–351
Bupivacaine HCL	<i>n</i> -Heptane/water 27.5	<i>Neural Blockade in Clinical Anesthesia and Management of Pain</i> , Issue 494 by Michael Cousins, Phillip O. Bridenbaugh, Lippincott Williams and Wilkins 1998
Baclofen	0.1	Hayek et al. <i>Seminars in Pain Medicine</i> 2003;1(4):238–253
Fentanyl citrate	813	<i>Neural Blockade in Clinical Anesthesia and Management of Pain</i> , Issue 494 by Michael Cousins, Phillip O. Bridenbaugh, Lippincott Williams and Wilkins 1998
Sufentanil citrate	1788	<i>Neural Blockade in Clinical Anesthesia and Management of Pain</i> , Issue 494 by Michael Cousins, Phillip O. Bridenbaugh, Lippincott Williams and Wilkins 1998

Source: Reprinted with permission of Deer.⁵⁰

■ Technique

An implanted system for the delivery of IT medication consists of an implanted pump and catheter system, either a single catheter including the subarachnoid and subcutaneous portions or a two-piece catheter system including the subarachnoid portion and separate subcutaneous portion connected with an implanted connector. The choice of catheter system is determined by the preference of the implanter. Prior to placement of a permanent catheter and pump system a trial for IT delivery of either baclofen or analgesic medication is usually

but not always performed. Trials for IT delivery of agents can be performed by single-shot CSF delivery of the agent, by continuous infusion of the agent via a temporary and externalized IT catheter, or by delivery of the agent through a permanent and internalized catheter subcutaneously connected to an externalized catheter and external pump.²⁸ There is no agreement as to how a trial should be performed; however, a recent expert panel recommends the following algorithm for trialing of patients for IT therapies, and also states that not all patients need to undergo a trial of IT therapy. These authors state, “trialing may not be appropriate for all patients. In some cases, socioeconomic factors may be involved in the decision of whether to forgo trialing. Furthermore, time constraints associated with terminal illnesses, such as cancer, can make trialing counterproductive and unnecessary. Trialing may also be unnecessary and/or detrimental in some nonmalignant conditions, such as cerebral palsy, or in patients who have had a stroke” (see **Fig. 8.1**).

Today there are several options for programmable pumps and catheters approved by the FDA, including Medtronic, Inc.’s (Minneapolis, MN, USA) SynchroMed II pump, Flowonix’s Prometra pump (Mt. Olive, NJ, USA), and Codman’s Medstream pump (Depuy Orthopedics, Warsaw, IN, USA) (see **Fig. 8.2**).

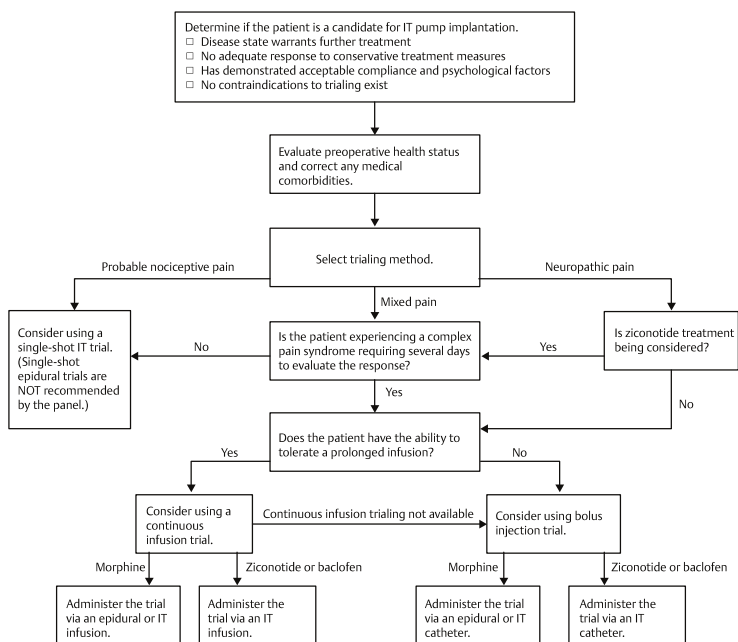


Fig. 8.1 Trialing algorithm for IT (intrathecal) analgesia. (Reprinted with permission of Deer et al.²⁸)



Fig. 8.2 Implantable programmable pumps. (a) Prometra from Flowonix. (b) Medstream (courtesy of Codman Neuro). (c) SynchroMed II by Medtronic (reprinted with the permission of Medtronic, Inc. ©2012).

Positioning and Surgical Preparation of the Patient for Catheter Placement

Preoperative antibiotics covering skin flora are recommended for the prevention of perioperative infections.²⁹ If the trial for IT therapy was performed either by single-shot injection of an agent, continuous infusion of agent(s) using a temporary catheter, or continuous infusion of agent(s) using a permanent catheter, the patient, after induction of general anesthesia or regional anesthesia—depending on the preference of surgeon, the patient, or both—is placed in either the left or right lateral decubitus position for placement of the IT catheter and implantable pump, with the intended pump site toward the ceiling of the operating room. An axillary roll is placed to relieve traction and pressure on the brachial plexus, and padding is placed under and between all bony protuberances. The arms are placed away from the surgical sites. The authors use the “praying mantis” position of the arms with a pillow between the upper and lower arms, paying careful attention to leave the endotracheal tube free for observation and emergency. Padding is placed beneath the lower knee and ankle, as well as between the legs. The patient is then secured to the table using either wide cloth tape or a “bean bag” security system. Either way, security must not encroach upon the surgical site.

Once secured to the table in the appropriate position, the patient is prepped and draped according to the customary process of the operating room and surgeon. Placement of the catheter system requires intraoperative fluoroscopy for both safety of the patient and to aid in identifying bony landmarks for catheter placement. Surgeons and operative personnel should take appropriate and recommended precautions for use of fluoroscopy. Using fluoroscopy, before prepping the patient, the skin is marked for intraoperative placement of incision for catheter placement and pump placement.

If the trial used a temporary catheter, then that catheter should have been removed prior to the permanent operation and a new incision made in the midline or paramedian to the vertebral body, the apex of the incision being one level below the level of the dura that the needle will enter. If the trial used a permanent catheter, then the externalized portion of the trial catheter should be “prepped” out of the sterile surgical field. The surgical site for placement of the trial catheter

is then opened to reveal the connection between the internalized and externalized catheter. The two catheters are disconnected and the internalized catheter end is clamped to prevent loss of CSF. The externalized catheter is gently pulled by a nonscrubbed staff member from under the drapes and discarded. The incision should be made large enough for anchoring of the catheter and carried down to the supraspinous fascia. A pocket is created by undermining the edges.

Care must be taken not to place the needle intraoperatively over the spinal cord to prevent needle placement into the spinal cord. If circumstance requires placement of an IT catheter directly over the spinal cord, then safety requires that the catheter be placed directly through a minilaminotomy and visualization of the dura and cord. A narrow, greater than 60-degree angle, using a paramedian approach of the 17-gauge epidural needle, is recommended for ease of catheter placement and advancement and to prevent kinking and damage to the catheter within the IT space (see **Fig. 8.3**). **Fig. 8.4** shows a fluoroscopic technique introduced

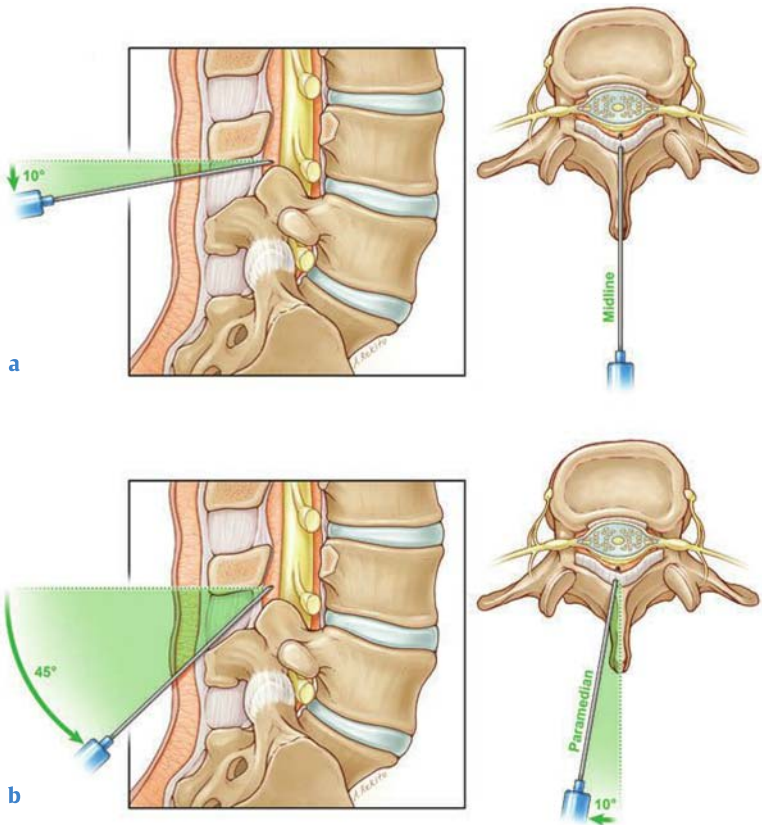


Fig. 8.3 (a, b) Paramedian approach to spinal canal.

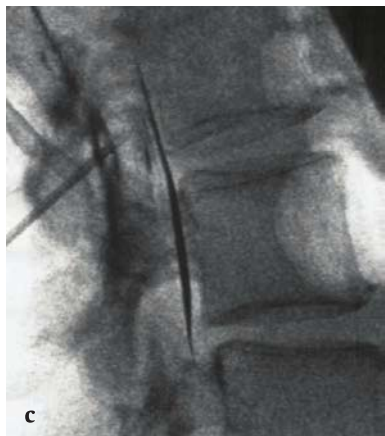
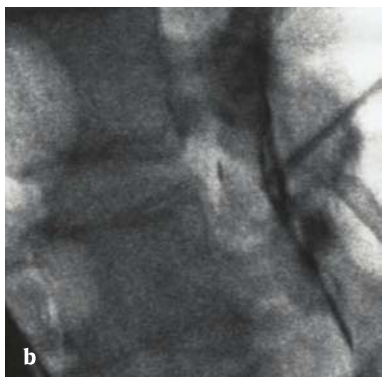
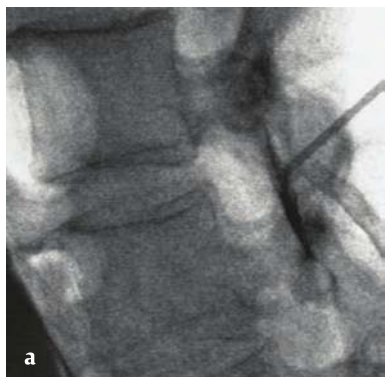


Fig. 8.4 (a) The epidural needle enters the epidural space using a loss-of-resistance technique and 0.5 mL of contrast dye is injected. Dye is seen layering out along the thecal sac. (b) The epidural needle is slowly advanced against the resistance of the dura mater. Dye is now seen tented along the dura mater and along the subdural membrane. (c) The needle is advanced further until there is a discernible loss of resistance or feeling of a “pop,” and dye is again injected. Seen posteriorly is the tenting of the dura and subdural membranes and anteriorly is the layering of dye along the anterior thecal sac. Confirmation of placement is made by observing a free flow of CSF. (From Haddadan and Krames,⁵⁸ Figs. 1, 2, and 3, John Wiley & Sons Ltd., reproduced with permission.)

by Haddadan and Krames to prevent multiple attempts and failures of placement of the IT catheter.

Under fluoroscopic guidance, the catheter is threaded through the needle to the desired spinal level, determined by pain complaint and lipophilicity of agent used (see **Table 8.1**). The catheter should pass easily through the needle. If resistance to advancement is encountered, the catheter should be carefully retracted *slowly* through the needle to prevent injury or tearing of the catheter. If resistance to pulling the catheter back is encountered and the catheter is not in optimal position, the surgeon should weigh the risks and benefits of removing and replacing the entire catheter and needle, with full awareness that removal means an unnecessary hole in the dura with its attendant complications of dural leak.

There is no firm agreement as to where the catheter tip should be placed; however, Krames recommends that the catheter be placed as close to the area of the

spinal cord that is processing the patient's pain as possible³⁰ to allow for the use of both lipophilic and hydrophilic agents (see **Table 8.1**). Good rules for catheter tip placement are that the catheter tip should be placed midspine for generalized pain as in multiple metastases from cancer pain, at the area of the spinal cord that is processing a single pain site, high in the cervical spine for head and neck pain, at midthoracic spine for spasticity of the lower extremities, high in the cervical spine for spasticity due to quadriparesis, and high in the cervical spine for dystonia.³¹

Once the catheter tip is placed in the optimal position, the needle is removed and the catheter is anchored to the fascia using the anchor of choice for the catheter selected by the surgeon. To prevent inadvertent removal of the catheter a strain loop should be created between the anchor and the pump in the pocket used for placement of the IT catheter. The authors use a permanent catheter trial; therefore, all old concentration of drug used during the trial is allowed to drain from the end of the catheter by allowing backflow of CSF. This usually amounts to less than 0.25 mL. The end of the catheter is again clamped to prevent further CSF leak.

Pump Placement

The pump is placed subcutaneously in the upper quadrant of the abdomen into a surgically created pocket and should be away from the iliac crest, the bladder, and previous incision sites. The choice of site for the pump, size of the pump, and cosmetic concerns should be discussed with the patient before surgery. There should be no surprises for the patient. The incision and created pocket should be just large enough for the pump of choice and no larger. These authors choose to use a pump with suture loops so that the pump can be anchored to the fascia within the pocket.

Once the pocket is created, a single-piece catheter is tunneled from the back incision to the pump pocket either using one pass in thin patients or via an intervening small, midflank stab wound in heavier patients. A provided catheter passer is used. If a two-piece catheter system is used, then the subcutaneous portion of the catheter system is tunneled from the pump pocket to the back pocket and connected to the IT portion of the system using a provided-for connector and strain relief by the manufacturer used. The authors allow CSF to drain to the end of the entire catheter system before connecting to the pump. The catheter system is then connected to the prepared (to manufacturer's specifications) pump before placement into the pocket.

Once the catheter is secured to the pump and the pump is placed into its pocket and the strain loop is created within the back pocket, both pockets are copiously irrigated with a sterile saline antibiotic solution and closed to the surgeon's specifications for closure. The incisions are appropriately dressed before leaving the operating room.

At this juncture, attention must be made to programming the pump, with consideration of CSF dead space within the catheter system according to recommendations of the manufacturer of the pump (see **Fig. 8.5**).

Risk Management and Complications of IT Therapy

Intraoperative risks include bleeding, damage to nerves and nerve rootlets, spinal cord damage and attendant neurologic sequelae, including paresis, bowel and bladder dysfunction, paralysis, neuropathic pain (including complex region-



Fig. 8.5 A pump within its abdominal pocket and catheter tunneled to the intrathecal spinal space.

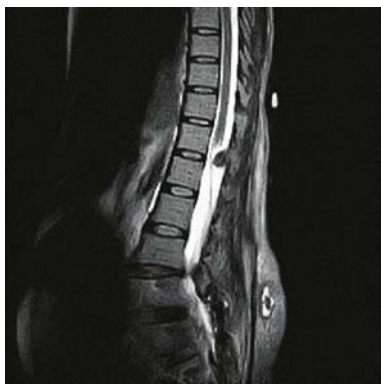


Fig. 8.6 Presence of IT granuloma. (Reprinted with permission of Deer et al.⁴⁶)

al pain syndrome [CRPS]), postspinal headache, blindness, deafness, epidural hematoma, subdural hematoma,³² epidural abscess, meningitis, wound infection, new back pain, and even death. These complications and risks should be discussed with the patient before proceeding with a trial, and informed consent should be obtained.

Intraoperative management should be performed with the intent of minimizing the risk of intraoperative complications and postoperative complications of the implanted system and delivery of drugs, including programming complications, development of IT granuloma and neurologic sequelae of the granuloma, myelitis, and respiratory complications. For a more in-depth discussion of the problems and management of the risks and complications of IT therapy, see Follett et al,²⁹ Naumann et al,³³ Deer et al,³⁴ and Taira et al.³⁵

The most-feared postimplantation complication of IT analgesic infusion is the development of IT granuloma, an intraspinal and nonmedullary granulomatous mass (see **Fig. 8.6**). Granulomatous masses, almost always at the tip of implanted IT catheters, are related to high doses and high concentrations of analgesic opioids, including morphine and other, nonmorphine opioids^{36,37}; are found in large animals (e.g., dogs, sheep) and humans; are characterized as distinct, globular, or spheroid collections of macrophages, plasma cells, eosinophils, or lymphocytes^{38–43}; are aseptic⁴⁴ as defined by staining or culture; and almost always arise from the arachnoid layer of the meninges and not from neuronal tissue of the spinal parenchyma. IT opioid-induced granulomas are not dependent on opioid receptor activation.⁴⁵ Instead, migration of inflammatory cells, most likely mast

cells, from the local meningeal vasculature appears to be an important component of granuloma formation.²⁰ For a more in-depth discussion of the diagnosis and management of IT tip granuloma, refer to the recommendations regarding IT granulomas from the Polyanalgesic Conference 2012.⁴⁶

The signs and symptoms of IT granuloma are listed in the accompanying box.

Signs and Symptoms Associated with Granuloma

- New or different sensory symptoms (e.g., numbness, tingling, burning, hyperesthesia, hyperalgesia, hypohesia, anesthesia)
- New occasional or intermittent bowel or bladder sphincter dysfunction
- New motor weakness, change in gait, or difficulty walking
- Any neurologic symptoms or signs that differ from baseline (e.g., reflex changes, clonus)
- Change in the character, quality, or intensity of pain
- The need for frequent or large escalations of the daily drug dose to maintain the analgesic effect
- Only temporary alleviation of increasing pain after rapid dose escalations
- Reports of new radicular pain, especially at or near the dermatomal level of the catheter tip

(Reproduced with permission of Deer et al.⁴⁶)

The recommendations of this panel for the prevention and detection of IT granuloma is presented in the next box. A treatment algorithm for IT granuloma is presented in **Fig. 8.7**. Present recommendations for dose and concentration limits are presented in **Table 8.2**.

Prevention and Screening for IT Granuloma

Prevention

1. Use the lowest effective concentration and dose of IT opioid agents, especially of morphine sulfate.
2. Use bolus dosing instead of continuous infusion for IT drug administration.
3. Consider placing the tip of the intraspinal catheter in the lumbar thecal sac, below the conus medullaris.
4. Implement adjuvant therapy with nonopioid analgesics if concerned about granuloma formation.
5. Switch from IT opioid therapy to ziconotide if concerned about a recurrence of granuloma.

Screening and Detection

1. Take a patient history and perform physical examinations on patients receiving IT opioid or baclofen therapy at least every 3 months.
2. Routinely monitor patients receiving opioids or baclofen for prodromal clinical signs or symptoms of granuloma.
3. Monitor the yearly rate of increase in drug dose.
4. Educate clinicians and radiologists about the radiological signs of granuloma.

(Reproduced with permission of Deer et al.⁴⁶)

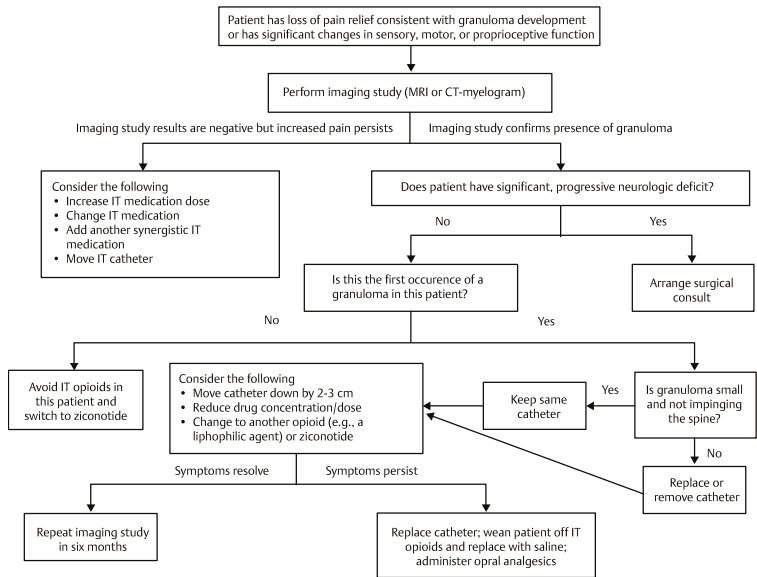


Fig. 8.7 Treatment algorithm for IT granuloma. MRI, magnetic resonance imaging; CT, computed tomography; IT, intrathecal. (Reprinted with permission of Deer et al.⁴⁶)

Table 8.2 Recommended maximum concentration and dose of IT agents to prevent IT granuloma

Drug	Maximum concentration	Maximum dose per day
Morphine	20 mg/mL	15 mg
Hydromorphone	15 mg/mL	10
Fentanyl	10 mg/mL	No known upper limit
Sufentanil	5 mg/mL	No known upper limit
Bupivacaine	30 mg/mL	10
Clonidine	1000 µg/mL	30–600 µg/d
Ziconotide	100 µg/mL	19.2 µg/d

Source: Reprinted with permission of Deer et al.⁵⁰

■ Indications for IT Therapy

In 1993, because there were no published studies comparing systemic administration of opioids with the IT delivery of opioids, Krames stated that patients with nonmalignant pain or cancer pain who failed appropriate long-acting

systemic administration of oral opioids (too many side effects) and who had greater than 50% reduction of pain during a trial of IT analgesic agents were candidates for a permanent IT administration as long as there were no contraindications, including psychological barriers, bleeding disorder, and failure to understand the therapy.³⁰ However, in 2002 Smith et al⁴⁷ performed a multi-center study comparing conventional medical management (CMM) of cancer pain with IT therapy of analgesic medication through and implantable drug delivery system (IDDS) for cancer pain. Sixty of 71 IDDS patients (84.5%) achieved clinical success compared with 51 of 72 CMM patients (70.8%, $p = 0.05$). IDDS patients more often achieved >20% reduction in both pain VAS (verbal analogue scale) and toxicity (57.7% [41 of 71] vs. 37.5% [27 of 72], $p = 0.02$). The mean CMM VAS score fell from 7.81 to 4.76 (39% reduction); for the IDDS group, the scores fell from 7.57 to 3.67 (52% reduction, $p = 0.055$). The mean CMM toxicity scores fell from 6.36 to 5.27 (17% reduction); for the IDDS group, the toxicity scores fell from 7.22 to 3.59 (50% reduction, $p = 0.004$). The IDDS group had significant reductions in fatigue and depressed level of consciousness ($p < 0.05$). IDDS patients had improved survival, with 53.9% alive at 6 months compared with 37.2% of the CMM group ($p = 0.06$). The authors concluded that IDDSs improved clinical success in pain control, reduced pain, significantly relieved common drug toxicities, and improved survival in patients with refractory cancer pain compared with CMM.

Today decisions on who is indicated for IT therapy must be made by the physician and patient, considering recommendations from the literature and reimbursement protocols of third parties. It is clear that good outcomes depend on good patient selection, and good patient selection should start with an appropriate psychological workup of the patient (see **Fig. 8.8**).



Fig. 8.8 Algorithm for psychological evaluation of patients for IT therapy. (Reprinted with permission of Deer et al.⁵⁰)

■ Opioid and Nonopioid IT Analgesia for the Control of Pain

Common chronic, nonmalignant disease states and diagnoses for which IT drug delivery is indicated include neuropathic pain syndromes (e.g., thalamic syndrome, spinal cord injury, diabetic neuropathy, postherpetic neuralgia), spinal stenosis, radicular pain from failed back surgery syndrome, complex regional pain syndrome, osteoporosis, compression fractures, pancreatitis, phantom limb pain syndrome, and other disorders caused by injury or irritation to the nervous system.⁴⁸ Unlike pain associated with terminal illness, noncancer pain patients tend to have longer life spans and require extended therapy that can range from months to years.⁴⁹

Present IT analgesics approved by the FDA include morphine and ziconotide (Prialt) and the antispasmodic medication baclofen for central spasticity. Other analgesic opioid agents that have been used include fentanyl, sufentanil, methadone, hydromorphone, and meperidine/pethidine. Nonopioid analgesic medications used for IT therapy include ziconotide, the local anesthetics, bupivacaine, lidocaine, ropivacaine and tetracaine, the α_2 -agonist clonidine, ketamine, droperidol, baclofen, gabapentin, midazolam, and various others. Agents that have shown neural toxicity and are not recommended for IT use include meperidine, methadone, tramadol, tetracaine, lidocaine, dexmedetomidine, and *N*-methyl-D-aspartate (NMDA) antagonists including ketamine, droperidol, midazolam, methylprednisolone, and ondansetron.²⁷ For a more complete discussion of this topic see Deer et al⁵⁰ and Penhollow et al.⁴⁸

Opioids administered neuraxially act at receptors in the substantia gelatinosa of the spinal cord dorsal horn to yield dose-dependent analgesia. Opioids may act through multiple mechanisms, including inhibition of presynaptic neurotransmitter release from primary afferents via presynaptic inhibition of calcium channels. Furthermore, opening of G protein gates, K⁺ channels in the central nervous system (G protein-regulated inwardly rectifying K⁺ channels [GIRKs]) may lead to postsynaptic neuronal hyperpolarization.^{48,51}

Ziconotide is an *N*-type calcium channel antagonist that is effective for the treatment of neuropathic, nociceptive, and mixed neuropathic–nociceptive pain. It is a synthetic derivative of a short peptide extracted from the venom of predatory cone snails (*Conus geographicus*, *Conus magus*) and is a member of a newly described chemical family called the conopeptides. Ziconotide blocks the *N*-type calcium channel located on the presynaptic terminal of dorsal horn C fibers. By blocking calcium entry into the presynaptic nerve terminal, ziconotide prevents the release of neurotransmitters into the synapse. Oxidized ziconotide is less active than ziconotide, so a pump containing a combination of ziconotide and another medication may need to be refilled more often. For this reason, the package insert for ziconotide does not recommend using the drug in combination with other drugs. However, a vast amount of clinical experience is surfacing on the use of ziconotide in combination with a variety of drugs with no adverse events reported.^{48,50,52}

Baclofen reduces spasticity and spasms by binding to gamma-aminobutyric acid (GABA_B) receptors and inhibiting the release of excitatory neurotransmitters, thereby inhibiting monosynaptic and polysynaptic spinal reflexes.¹⁵ Baclofen is not taken up into the brain tissue GABA system, and very little reaches the fourth ventricle to have any effect on respiratory centers.⁵³ Instead, IT baclofen has a selective effect on spinal neurons, which control reflex excitation of motor neurons, sparing other sensory and motor input.^{54,55}

■ Present Recommendations for the Rational Use of IT Polyanalgesia

There have been four polyanalgesic conference recommendations for the rational use of IT agents based on a review of evidence from the literature and a consensus of expert opinion regarding the topic.^{5,50,56,57} The present recommendations for the rational use of IT agents were presented in Polyanalgesic Conference 2012.⁵⁰ This panel of experts recommended different pathways for neuropathic and nociceptive pain with special considerations for cancer pain. Some recommendations have already been acknowledged. **Tables 8.3** and **8.4** outline the present recommendations for use of analgesic agents.⁵⁰

Table 8.3 2012 polyanalgesic algorithm for IT therapies for nociceptive pain

Line 1	Morphine, ziconotide, morphine + bupivacaine
Line 2	Hydromorphone + bupivacaine, morphine or hydromorphone + clonidine
Line 3	Clonidine, ziconotide + opioid, fentanyl, fentanyl + bupivacaine or fentanyl + clonidine
Line 4	Opioid + clonidine + bupivacaine, bupivacaine + clonidine
Line 5	Baclofen

Line 1: Morphine and ziconotide are approved by the U.S. Food and Drug Administration (FDA) for IT therapy and are recommended as first-line therapy for nociceptive pain. Hydromorphone is recommended on the basis of widespread clinical use and apparent safety. Fentanyl has been upgraded to first-line use by the consensus conference.

Line 2: Bupivacaine in combination with morphine, hydromorphone, or fentanyl is recommended. Alternatively, the combination of ziconotide and an opioid drug can be employed.

Line 3: Recommendations include clonidine plus an opioid (i.e., morphine, hydromorphone, or fentanyl) or sufentanil monotherapy.

Line 4: The triple combination of an opioid, clonidine, and bupivacaine is recommended. An alternative recommendation is sufentanil in combination with either bupivacaine or clonidine.

Line 5: The triple combination of sufentanil, bupivacaine, and clonidine is suggested.

Source: Reprinted with permission of Deer et al.⁵⁰

Table 8.4 2012 polyanalgesic algorithm for IT therapies for neuropathic pain

Line 1	Morphine, ziconotide, morphine + bupivacaine
Line 2	Hydromorphone + bupivacaine, morphine or hydromorphone + clonidine
Line 3	Clonidine, ziconotide + opioid, fentanyl, fentanyl + bupivacaine or fentanyl + clonidine
Line 4	Opioid + clonidine + bupivacaine, bupivacaine + clonidine
Line 5	Baclofen

Line 1: Morphine and ziconotide are approved by the U.S. Food and Drug Administration (FDA) for IT therapy and are recommended as first-line therapy for neuropathic pain. The combination of morphine and bupivacaine is recommended for neuropathic pain on the basis of clinical use and apparent safety.

Line 2: Hydromorphone, alone or in combination with bupivacaine or clonidine, is recommended. Alternatively, the combination of morphine and clonidine may be used.

Line 3: Third-line recommendations for neuropathic pain include clonidine, ziconotide plus an opioid, and fentanyl alone or in combination with bupivacaine or clonidine.

Line 4: The combination of bupivacaine and clonidine (with or without an opioid drug) is recommended.

Line 5: Baclofen is recommended on the basis of safety, although reports of efficacy are limited.

Source: Reprinted with permission of Deer et al.⁵⁰

■ Conclusion

In this chapter we present the relevant anatomy of the spinal canal as it pertains to IT therapies, the indications for IT analgesia, the techniques of implantation, the risks and complications of IT analgesia, and up-to-date recommendations for the rational use of analgesic medications via the IT space. This chapter is meant as a short overview, and the reader is welcome to delve into the relevant literature of IT analgesia. Present knowledge is summarized by the Polyanalgesic Conference 2012 and the recent updates of prior conferences 2000, 2004, and 2007.

Editor's Comments

The use of intrathecal (IT) analgesic therapy has waned over the past few years, to the extent that few neurosurgeons are engaged in this activity. Despite this, the possibility of IT analgesia should still be entertained in select diagnoses. Although IT therapy is probably underutilized for pain related to cancer, most common clinical indications are in the domain of noncancer pain.

The indications for IT therapy vary with the agent infused. Opiates (morphine, hydromorphone, fentanyl, sufentanil) are generally recommended for nociceptive pain, whereas nonopiates such as clonidine and ziconotide would be preferable for neuropathic pain. Mixing agents within the pump reservoir, particularly at the inception of therapy, has the disadvantage of locking in a medley of drugs with a fixed concentration ratio; this leaves no possibility of altering the administration of one drug while holding another constant, at least until a new admixture can be injected into the pump reservoir.

(Continued)

Editor's Comments (*Continued*)

The method of intrathecal trial is one area of variability in practice, as mentioned in this chapter. In my opinion, the potential for an intrathecal trial is one of the major advantages of this therapy, and should be conducted in every case. Our practice has been to use a "single-shot" trial for opiates, and this is certainly more expeditious than a prolonged intrathecal or epidural catheter trial, which requires hospitalization.

Intrathecal analgesic administration is a complex therapy, and it is easy to underestimate the potential for problems. For example, intrathecal opiate administration is almost universally accompanied by endocrinopathy in men and women, with decreased gonadotropins in both genders and, in particular, decreased testosterone in men. Withdrawal from intrathecal opiates can occur for a number of reasons, including pump or catheter malfunction, which occurs at a 10% rate every year. Intrathecal opiate withdrawal is painful and distressing but rarely fatal. Baclofen has been used intrathecally for pain control, as the authors mention, and withdrawal from this agent *has* been occasionally fatal.

Finally, every neurosurgeon should be familiar with the potential for catheter granuloma formation with IT opiate administration. This seems to be a sterile chemical irritation of the leptomeninges, related to the *concentration* of the delivered agent, particularly when the opiate is given in the range of hundreds of milligrams per day. Once a focal arachnoiditis develops, the distribution of the infused drug becomes even more limited, and the local concentration rapidly escalates in a regenerative process producing more chemical meningitis, and even more limited drug distribution, eventually leading to a frank granuloma. Drug concentration does appear to be the culprit, since delivery of drugs at levels of hundreds of micrograms per day (e.g., baclofen) essentially never produces catheter granulomas.

The authors outline the hallmarks of the development of a catheter granuloma. Neurosurgeons should know that once a diagnosis of a catheter granuloma has been made by MRI (magnetic resonance imaging), a complete resection is extremely ill-advised. Most of these granulomas occur in the thoracic area, where CSF (cerebrospinal fluid) flow is limited, and low-flow ("stagnant") areas are common. If the granuloma is producing a neurologic deficit, it can be decompressed by debulking, but should not be dissected off of the spinal cord or nerve roots. This can result in a severe neurologic deficit. Smaller, and minimally symptomatic, granulomas can be treated by simple withdrawal of the catheter from the granuloma, with or without replacement of the catheter at a different location. The granuloma will then regress, and the spinal cord will be decompressed. Due to the volume and flow of CSF in the lumbar theca, placement of the catheter in this area either primarily, if possible, or during catheter revision has a very low incidence of granuloma formation.

Intrathecal drug administration still represents a unique portal of entry into the blood-brain barrier, one that obviates both systematic parenteral administration and the enteral route. It is likely that some forms of advanced analgesic therapy will be able to leverage this modality to produce clinically significant analgesia. Neurosurgeons should continue to be involved in these procedures, particularly as the technology and pharmacology improve.

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9

Intrathecal Drug Administration: Patient Selection and Implant Technique

Daryoush Tavanaiepour and Robert M. Levy

As with most interventional therapies for chronic pain, in the administration of intrathecal opioids, careful patient selection appears to be the greatest and possibly the only significant predictor of success.

The precise variables that predict the success or failure of intrathecal opioids for the treatment of intractable pain remain poorly defined. Several factors that may indicate or contraindicate intraspinal analgesic treatment must be taken into account, and these are outlined in the accompanying box. In clinical practice, it is a valuable exercise to review these factors systematically for each patient before the decision is made to embark on chronic intrathecal opioid therapy.

■ Indications

Physiological Source of Pain

Prior to a trial of intraspinal opioids, it is important to ensure that a full diagnostic evaluation has been performed and that a cause for the pain has been established. If no reasonable cause for the pain is apparent after a careful history and physical examination have been performed, the probability of a significant functional component to the patient's pain becomes great, and the likelihood of long-term pain relief with intraspinal opioids falls significantly.

It is important to perform a careful pain diagnostic evaluation because many pain problems referred to the pain specialist are in fact treatable without the use of chronic interventional techniques. First, correctable causes of chronic pain must be established; direct therapy may result in pain relief. Thus, patients suffering from unrecognized peripheral nerve entrapment syndromes may have profound relief with decompression and transposition procedures; patients with neuromatous pain may benefit from neuroma excision and nerve stump implantation; and patients with significant spinal pathology may benefit from decompression or fusion procedures.

In addition to careful history taking, several features of the physical examination can aid in the diagnosis of chronic pain syndromes. Not only is it important to recognize and map regions of objective or subjective sensory loss, but it is also important to recognize abnormal sensory phenomena that accompany specific chronic pain syndromes. Thus, *allodynia*, the interpretation of a light touch stimulus as being painful, and *hyperpathia*, the interpretation of a mildly noxious stimulus as being significantly painful, are often hallmarks of neuropathic pain syndromes. The careful examination of skin temperature, sweating patterns, capillary filling time, hair growth, and possible trophic changes can indicate the

Patient Selection Criteria for Chronic Intraspinal Opioid Administration	
Absolute Indications • Known physiological source of pain • Failure of maximal medical therapy • Favorable psychosocial evaluation • Favorable response to intraspinal opioid trial	Absolute Contraindications • Intercurrent infection • Uncorrectable coagulopathy • Allergy to agent to be infused
Relative Indications • Nociceptive pain syndromes • Axial/multifocal/diffuse pain syndromes	Relative Contraindications • Intractable side effects • Obstruction to cerebrospinal flow

presence of a sympathetically maintained pain syndrome. The presence of focal muscle spasm with predictable patterns of referred pain on palpation may indicate a myofascial pain syndrome.

In the course of diagnostic evaluation, great value can be obtained from the use of specific local nerve blockade. It is vitally important for the surgeon either to perform the blocks or to know well the techniques used by the anesthesiologist performing the nerve blockade. By the use of small volumes of properly placed local anesthetics, the physician often can determine not only what nerves are involved in the perpetuation of the chronic pain syndrome, but also whether the somatic or sympathetic nervous system is involved and whether the pain is entirely peripheral or has a central component. These features can be remarkably important in predicting the efficacy of interventional pain procedures.

Numerous nonsurgically treated conditions are often overlooked by physicians referring patients for interventional pain therapy. Most commonly identified are myofascial pain syndromes, which may respond significantly to trigger point injections with local anesthetic, followed immediately by myofascial physical therapy directed at the affected muscle groups. Once the myofascial pain problem is appropriately treated, the residual chronic pain syndrome can be better characterized and more specifically addressed.

Failure of Maximal Medical Therapy

If a noninvasive regimen of analgesics provides satisfactory pain relief without intolerable side effects, intraspinal drug administration is not necessary. The World Health Organization (WHO) has published and widely distributed its schema for the management of pain secondary to malignancy.¹ Thus, patients with pain of malignant origin are seldom referred unless such a regimen has failed.

Failure to respect this patient selection criterion becomes an issue most often in the setting of pain of nonmalignant origin. Particularly relevant prior to the consideration of chronic intraspinal opioids is the consideration of the use of chronic oral or transdermal narcotics for pain of nonmalignant origin. Recommendations by the American Pain Society and the American Academy of Pain Medicine include the careful, regulated use of chronic narcotics for patients with pain of non-malignant origin.² Thus, if oral or transdermal administration provides adequate pain relief without unacceptable side effects, intraspinal drug administration is

not indicated. In routine clinical practice, oral or transdermal narcotics are given in a dose escalation paradigm until the patient achieves adequate pain relief or develops unacceptable side effects.

Thus, intraspinal narcotics should be reserved for patients in whom oral or transdermal narcotics either provide inadequate pain relief or produce truly unacceptable or uncontrollable side effects.

Favorable Psychosocial Evaluation

Although most investigators have highlighted the importance of a favorable psychosocial evaluation in the screening of potential implant candidates, wide agreement has not been reached in regard to the specific variables, their quantitation, and treatment. As part of this evaluation, most agree that both the patient and the patient's psychological and social support systems need to be evaluated. Clearly, acute psychotic illnesses and severe, untreated depression need to be diagnosed and effectively treated before consideration of surgery.

The presence or absence of medicolegal complications, such as worker's compensation or litigation, does not appear to predict outcome. Similarly, the number of prior pain-directed surgeries, the length of the chronic pain syndrome, the time away from work, or the patient's age or gender do not have clear predictive value.³

Pain Type: Predominance of Nociceptive Pain

Certain features of pain have been suggested as predictive of the efficacy of intraspinal morphine therapy. Intraspinal opioids are particularly effective in the presence of predominantly nociceptive chronic pain syndromes. Arner and Arner⁴ placed in rank order the types of pain responding to epidural morphine administration in their experience. This ranking is presented in the designated box.

Whereas neuropathic pain syndromes may require higher doses of intrathecal opioids and ultimately may be better treated with nonopioid intraspinal analgesics, the presence of neuropathic pain is not an absolute contraindication for evaluating intraspinal opioid therapy.

Pain Distribution: Predominance of Axial/Diffuse Pain

For axial pain syndromes, especially those involving the low back or neck, or for multifocal intractable pain syndromes, intraspinal opioids are particularly effective and should be considered. Regarding appendicular neuropathic pain states, spinal cord stimulation is particularly effective in this setting. Only after spinal cord stimulation has been attempted and failed should intraspinal opioids be investigated for solely appendicular pain syndromes.

Ranking List of Different Cancer-Related Pain Types with Regard to the Likelihood of Relief with Epidural Morphine Treatment

1. Somatic, continuous
 2. Visceral, continuous
 3. Somatic, intermittent
 4. Visceral, intermittent
 5. Neurogenic, intermittent and continuous
 6. Cutaneous (cancerous ulcer or fistula)
- (From Arner and Arner.⁴)

Favorable Response to Intraspinal Opioid Trial

The response to acute intraspinal administration of analgesic agents is generally regarded as an excellent indicator of long-term efficacy.⁵ The inability to achieve pain relief after such a trial is a contraindication to implantation. Several approaches to the trial of intrathecal narcotics have been advocated, including single versus multiple injections, administration by lumbar puncture versus indwelling catheter, epidural versus intrathecal routes, and bolus versus continuous infusion administration of the drug. Continuous epidural or intrathecal infusions are preferred over bolus administration trials because they better reflect the dynamics of chronic drug infusion and may predict the effective dose.

Attempts to control for patient bias by testing both morphine and saline and blinding the patient to which drug is being infused still do not control for the bias of the health care team. Another significant drawback to many attempts at preimplantation intraspinal opioid trials is their lack of objective measures. This subjectivity can negatively impact the validity and reliability of screening protocols.

To address these concerns, a quantitative, crossover, double-blind trial for the preimplantation screening of candidates for chronic drug infusion therapy for the control of intractable pain has been developed.⁵ Application of this protocol resulted in the elimination of about 30% of potential implant candidates. Of patients whose screening trial was successful, about 70% have had good to excellent long-term pain relief. This screening paradigm appears to be both reliable and easily applied. Furthermore, the Polyanalgesic Consensus Conference (PACC) 2012, an expert panel of experienced physicians on intraspinal analgesics for chronic pain, has published a comprehensive analysis and algorithm for intraspinal opioid trials (see **Fig. 9.1** for details).⁶

■ Contraindications

Intercurrent Infection

Any local infection at the placement sites or the presence of systemic infection contraindicates the implantation of drug administration devices. A period of observation after completion of antibiotic therapy is recommended prior to the implantation of drug infusion devices. Furthermore, the use of perioperative and postoperative prophylactic antibiotics is recommended.

Uncorrectable Coagulopathies

Coagulopathies can complicate the procedure with the development of subcutaneous, epidural, or intradural hematomas. Significant uncorrectable coagulation disorders absolutely contraindicate the implantation of a drug infusion system.

Allergy to Infused Agent

Allergy to the analgesic agent to be infused obviously and absolutely contraindicates its use.

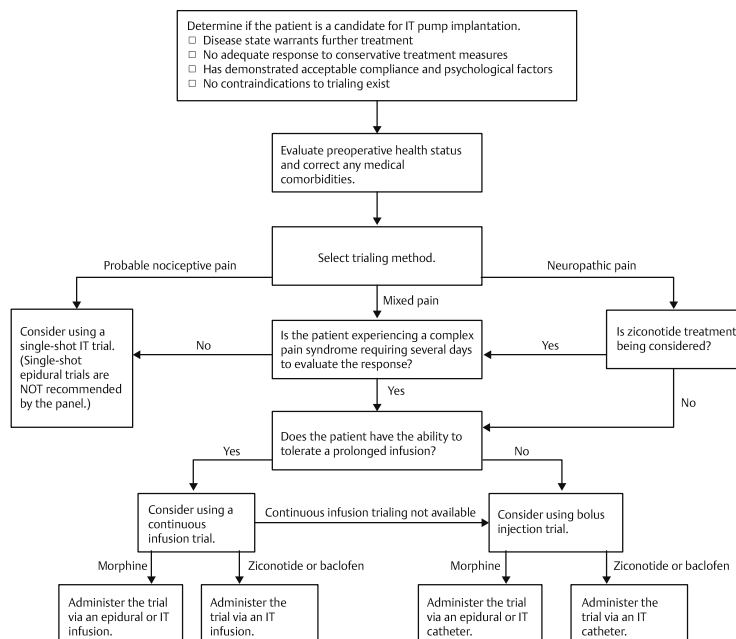


Fig. 9.1 The 2012 Polyanalgesic Consensus Conference algorithm on trialing for intrathecal (IT) drug delivery.⁶

Intractable Side Effects

The most widely recognized side effects of intraspinal narcotics include urinary retention, pruritis, and, rarely, delayed respiratory depression. During intraspinal opioid screening, particularly with bolus administration, the clinician may observe these side effects. They are, however, only relative contraindications to chronic drug administration. With the use of lower opioid doses and chronic administration, these side effects usually resolve. The potential pain relief achieved by intraspinal opioids must be balanced against the severity of potential side effects.

Obstruction of Cerebrospinal Fluid Flow

Obstruction of cerebrospinal fluid (CSF) flow has been suggested as a relative contraindication to intraspinal drug delivery, depending on the size, location, and cause of the obstruction. In our experience, this has not been a significant problem, and patients have received excellent drug effects despite the presence of significant degrees of CSF flow obstruction. This is probably because the usual site of pain generation is near the site of obstruction, so that sufficient CSF opioid concentrations are still reached at the target spinal cord site of pain transmission.

Severely Limited Life Expectancy

Whereas the expected length of life is not a contraindication to the intraspinal route of drug administration, it does bear greatly on the decision as to which method of administration to use. Thus, percutaneous epidural catheters attached to external pumps, internalized passive reservoirs and catheters requiring percutaneous drug administration, patient-activated mechanical systems, constant-rate infusion pumps, and programmable infusion pumps are all options for intraspinal drug delivery.

For patients who are largely bedridden and whose life expectancy is only weeks, the expense and risk of surgical pump implantation are not indicated. Rather, these patients are well treated with a tunneled epidural catheter.

In patients with short life expectancy who are ambulatory and in whom a tunneled epidural catheter would significantly affect mobility, a subcutaneous reservoir attached to an intrathecal catheter is an excellent option.

In patients with a life expectancy of greater than 3 months, implanted drug pumps become a viable option for intraspinal drug delivery. Nonprogrammable drug pumps, which deliver drug at a constant rate, are less expensive than programmable pumps and are well suited for patients in whom drug requirements are well defined and in whom frequent dose changes are not anticipated. For patients in whom tailored drug delivery regimens are desired or in whom frequent dose changes are anticipated, the additional expense of implanted programmable drug pumps is warranted.

■ Surgical Technique

A detailed description of the surgical procedure can be found elsewhere.^{7,8}

Preoperative Preparation

- Appropriate patient selection (see above)
- Pump preparation (programming, calibration, drug selection/preparation)
- Appropriate antibiotics
- Availability of fluoroscopy

Intraoperative Procedure

- *Patient positioning:* Lateral decubitus with right/left flank up (depending on abdominal pump insertion site), axillary role with pressure points padded
- *Pump pocket incision:* Lower right/left abdomen with consideration of iliac crest, ribcage, and patient's belt-line location. The incision should not directly overlay the final position of the pump. A subcutaneous pocket is fashioned above the rectus fascia large enough to accommodate the pump without undue tension to prevent wound dehiscence.
- *Lumbar incision:* Make a midline incision overlying the L3–4 region, approximately 4 cm in length with exposure of the lumbodorsal fascia. Develop a subcutaneous pocket sufficient to allow for anchoring and coiling of the catheter.
- *Insertion of intrathecal catheter:* Enter subarachnoid space with a Tuohy needle at a paramedian and shallow trajectory at the L2–3 or L3–4 level using

fluoroscopy. Confirm CSF flow and with the bevel cephalad, advance the intrathecal catheter. Verify catheter position with fluoroscopy. With the introducer needle still in position, place a pursestring suture in the fascia and two additional sutures on opposite sides of the needle for anchoring the catheter. Carefully remove the needle and guide wire simultaneously, secure the catheter with the anchor system, and tie the pursestring suture to close the fascia around the catheter.

- **Install the pump:** Tunnel the catheter and connect the intrathecal catheter to the pump. Allow for a relaxing loop of the intrathecal catheter at the spinal fascia to accommodate for patient movement and to prevent kinking. Any excess proximal catheter is coiled beneath the pump (to prevent injury during refill or future revisions). The pump is placed within the subcutaneous pocket with the access port facing the skin. The pump is secured to the abdominal fascia using nonabsorbable sutures. The wounds are copiously irrigated with antibiotic solution and closed in layers.

A set of comprehensive and detailed recommendations to reduce morbidity and mortality related to intrathecal drug delivery system has been provided by the 2012 PACC.^{9,10}

Editor's Comments

Intrathecal drug administration holds substantial promise for the surgical management of pain, particularly in the areas of cancer-related pain and neuropathic pain.

Although the frequency of use of intrathecal opiates for cancer-related pain is unknown, my impression is that this technique is used very little. If 70 to 90% of cancer patients achieve effective pain relief using the WHO recommendations, that leaves 10 to 30% who do not. The principles of “by the mouth,” “by the clock,” “by the ladder,” “for the individual,” and “attention to detail” are sound and humane. The indications for more aggressive pain control, be it intrathecal agents or ablative surgery, would be fulfilled by the failure of that standard approach—the next step on the ladder. If even 10% of cancer patients have uncontrolled pain, why is it that the administration of intrathecal opiates has been so underutilized? There seems to be a cultural divide between oncology and surgical pain management that, in my opinion, represents a major disconnect and a major failure in our treatment of cancer.

Intrathecal opiates are still being used for the management of chronic pain not related to cancer. Their use remains somewhat controversial, and as with many surgical procedures, high-quality data to support their use is lacking. The use of opiates delivered by pump and intrathecal catheter has dwindled in the past decade, due largely to the problems of escalating doses in many patients, complications of the therapy such as catheter granulomas, and decreased funding by Medicare and other payers. Without better data, it is likely that the use of intrathecal opiates for “benign” pain will disappear entirely.

The effective treatment of neuropathic pain has been the “holy grail” of pain medicine over my entire career. Unfortunately, the pathophysiology of neuropathic pain must involve such fundamental properties of the nervous system (e.g., memory) that it has not yet been possible to develop oral analgesic agents that both relieve neuropathic pain and do not produce intolerable side effects.

Dr. Levy outlines patient selection and implant strategies for intrathecal drug delivery systems. The use of “pumps” for pain control is an important aspect of interventional pain management, which will only be amplified when cancer pain is more aggressively treated, and the appropriate intrathecal agent for neuropathic pain is discovered.

■ Conclusion

At present, the data concerning intraspinal opioids for pain secondary to cancer appear to be compelling and consistent, indicating a success rate of 70 to 80%. Data concerning their use in the setting of pain of nonmalignant origin are less clear and consistent, although recent unpublished findings suggest a similar efficacy of 65 to 75%. By ensuring that patient selection criteria are rigorously adhered to, the clinician may hope to expect similar rewarding outcomes.

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Section II.D

Procedures for Craniofacial Pain

10

Surgical Options for Facial Pain

Konstantin V. Slavin and Kim J. Burchiel

Much has been written about various surgical procedures developed to treat pain in the face. The list is long; there are at least 10 well-established surgical interventions that are commonly (or not so commonly) used to treat facial pain patients. And since surgical interventions are traditionally reserved for those who have failed medical management, the mere presence of these surgeries indicates the relative ineffectiveness of conservative (less invasive) treatments due to either unresponsiveness of the underlying condition to medications, development of eventual tolerance to initially effective regimens, or a high incidence of side effects that occur during required dose escalations.

However, it is rather obvious that if any condition has this many (surgical) treatments, and each of the surgical procedures is both effective and safe, there must be some rationale for choosing one procedure over all others in each specific clinical case. Ideally, such a rationale should be supported by high-level scientific evidence, perhaps through multiple independent, prospective, randomized, controlled studies comparing treatment modalities and weighing the benefits of each intervention against its risks and potential failures. But despite decades of widespread clinical use of most of these surgeries, the desired evidence is either weak or nonexistent, and the support for each intervention comes from anecdotal observations, retrospective (albeit quite large) case series, and uncontrolled, non-randomized comparative evaluations.

Nevertheless, the main challenge in surgical management of facial pain is not in the overabundance of surgical approaches but in the matching of each individual patient with the most appropriate surgical intervention. The difficulty here lies in the wide variety of overlapping clinical syndromes that present with facial pain¹ and in the differential response of each syndrome to specific types of surgery. Therefore, any algorithm designed for the rational use of surgical interventions in the treatment of facial pain must start with establishing the correct diagnosis, and only after that may additional criteria be used to determine appropriate interventions and the order in which they should be offered to each patient. Ultimately, in those situations where more than one treatment option is available, the patient's preference should be taken into consideration, making the patient a part of the decision-making process.

Since our practical algorithm was published several years ago,² we have successfully applied it to facial pain patients presenting to us. Here we present an overview of surgical interventions and review the decision-making process used in selection of the appropriate approach.

■ Causes and Features of Facial Pain

Surgery for facial pain starts with establishment of the diagnosis, which may be the most difficult part of the entire treatment process. The face is the site of a wide variety of pain syndromes. Some are common, such as headaches and toothaches; some are relatively rare, like classic (typical) trigeminal neuralgia and post-traumatic trigeminal pain; and some are rarer still, for example, sphenopalatine neuralgia and glossopharyngeal neuralgia.

Central to the diagnosis of facial pain is obtaining a detailed history, with special attention to the character of pain, its distribution, triggering, the presence of remission, and response to medications. Unilateral sharp, lancinating pain limited to the territory of one or several divisions of the trigeminal nerve with short attacks and associated trigger points is characteristic of *idiopathic (typical) trigeminal neuralgia*. Other typical features of this condition are the absence of pain between attacks; frequent remissions, especially early in the course of the disease; normal neurologic examination; and a high degree of pain relief in response to oral carbamazepine.

Differential Diagnosis of Facial Pain	
Trigeminal neuralgia (tic douloureux)	• Genuiculate neuralgia
• Trigeminal neuralgia type I: idiopathic typical trigeminal neuralgia	• Sphenopalatine (Sluder) neuralgia
• Trigeminal neuralgia type II: idiopathic atypical trigeminal neuralgia	• Auriculotemporal neuralgia
• Secondary trigeminal neuralgia	• Nasociliary neuralgia
Trigeminal neuropathic pain	Paratrigeminal (Raeder) syndrome
• Posttraumatic trigeminal pain	Painful ophthalmoplegia (Tolosa–Hunt syndrome)
Trigeminal deafferentation pain	Petrous apex syndrome (Gradenigo syndrome)
• Anesthesia dolorosa	Cancer-related pain
• Central deafferentation syndromes	Atypical facial pain
– Wallenberg syndrome	Orofacial pain and temporomandibular joint-related pain
– Thalamic syndrome	Headache and migraine syndromes
Postherpetic neuralgia	
Other cranial neuralgias	
• Glossopharyngeal neuralgia	

The cause of the idiopathic trigeminal neuralgia remains unknown; however, a great majority of patients have compression of the trigeminal nerve root by adjacent vessels, most commonly by the superior cerebellar and the anterior inferior cerebellar arteries.^{3,4} This and the fact that pain was almost uniformly relieved after decompression of the root support the current belief in the importance of microvascular compression in the cause and pathogenesis of typical trigeminal neuralgia. The finding of focal demyelination in the intracranial trigeminal nerve root associated with prolonged vascular root compression further supports this theory and the surgical treatment of trigeminal neuralgia with microvascular decompression in the posterior fossa.⁵ If the facial pain has some features of typical trigeminal neuralgia but differs from its classic description by the presence of

hypesthesia in the trigeminal distribution, absence of response to carbamazepine, or additional constant pain that persists between classic attacks of the sharp, electric shocklike pain, then the diagnosis of *atypical trigeminal neuralgia* may be made.⁶

Typical and atypical trigeminal neuralgia may be difficult to differentiate since some patients may have features that change over time. As a matter of fact, the term *atypical trigeminal neuralgia*, indicating predominance or even the presence of a “permanent background of pain,”⁷ has been largely replaced by the term *trigeminal neuralgia type II* and the typical trigeminal neuralgia is now referred to as *trigeminal neuralgia type I*.^{1,8} Moreover, we theorized that trigeminal neuralgia may progress from type I to type II as the part of its natural history.⁹

The atypical features, especially sensory deficits and constant pain, should raise the possibility of the facial pain being a symptom of another pathologic process.

Causative Conditions of Secondary Trigeminal Neuralgia

Demyelinating disease

- Multiple sclerosis

Neoplasms

- Meningioma
- Trigeminal schwannoma
- Epidermoid
- Vestibular schwannoma
- Nasopharyngeal carcinoma
- Metastases
- Brainstem glioma

Vascular lesions

- Aneurysm
- Arteriovenous malformation
- Megadolichobasilar artery
- Persistent primitive trigeminal artery

Sarcoidosis

Connective tissue disorders

- Scleroderma
- Sharp disease (mixed connective tissue disease)

Syringobulbia

Pseudotumor cerebri

Paget disease

Acromegaly

Amyloidomas

Syphilis

Arnold–Chiari malformation

This is related primarily to various mass lesions in the posterior fossa (tumors and vascular malformations) or in the trigeminal ganglion and root that must be ruled out by using modern neuroimaging techniques, such as magnetic resonance imaging (MRI). Usually, the pain in these cases is referred to as *secondary trigeminal neuralgia*, although one may argue that even idiopathic trigeminal neuralgia technically may be considered a secondary symptom of the primary pathologic process of neurovascular compression. Treatment of the secondary trigeminal neuralgias usually is aimed at elimination of the underlying problem; the symptomatic pain may resolve completely once the mass lesion or other offending process is removed or otherwise treated.^{10,11}

If the neuroradiologic workup of trigeminal neuralgia type II does not show any underlying structural pathology, it is considered to be idiopathic. We believe that, in many instances, type II is a more severe or advanced form of typical, type I trigeminal neuralgia, and that it is seen in those cases in which vascular compression of the trigeminal nerve root is significant enough to produce not only mild demyelination with resulting paroxysmal pain but also a more overt neuropathy

with sensory loss and chronic constant pain. In our opinion, surgical exploration and microvascular decompression are the definitive treatment for this pain syndrome.^{7,9,12,13}

A syndrome resembling typical trigeminal neuralgia is encountered frequently in patients who have multiple sclerosis and in patients with brainstem infarction at the level of the trigeminal root entry zone.^{14–16} The features of this “symptomatic” trigeminal neuralgia do not differ from the typical form of the disease, but treatment focuses on neuroablative interruption of the trigeminal nerve, retro-gasserian root, or brainstem pathway rather than microvascular decompression.

Posttraumatic trigeminal pain or *trigeminal neuropathic pain* shares clinical features with other peripheral neuropathic pain syndromes; it is associated with constant dull, throbbing pain and paroxysms of sharp pain that may be burning in nature. The underlying mechanism of this type of pain is thought to be the injury of the trigeminal nerve or its branches, more distal than in cases presenting with typical trigeminal neuralgia.¹⁷ What is not clear, however, is whether any trigeminal neuromata or other peripheral deafferentation can be found. Treatment of the posttraumatic trigeminal pain usually follows the same therapeutic algorithm as the rest of peripheral neuropathic pain syndromes: If conservative management fails, surgery on the peripheral trigeminal system is considered, followed by peripheral trigeminal stimulation and then by more central surgical interventions (e.g., ganglion, root, brainstem).^{18–21}

As a part of the effort to classify trigeminal pain syndromes based on etiology with a focus on subsequent simplification and standardization of treatment paradigms, the term *trigeminal neuropathic pain* is reserved for cases of pain due to unintentional trigeminal injury, such as facial trauma, oral or otorhinolaryngological surgery, posterior fossa operations not aimed at the trigeminal nerve, or stroke; cases of pain due to intentional trigeminal nerve injury such as neurectomy, gangliolysis, rhizotomy, and other peripheral or central denervating procedures are referred to as *trigeminal deafferentation pain*.^{1,8}

Somewhat similar, but even more severe and frustrating, is pain associated with sensory loss in the face. This syndrome of *trigeminal anesthesia dolorosa* sometimes develops after brainstem and mesencephalic infarctions, but more commonly it is observed after destructive procedures on the trigeminal system that are used to treat trigeminal neuralgia, such as open or percutaneous rhizotomy, neurectomy, or tractotomy. In essence, trigeminal anesthesia dolorosa is an extreme case of either trigeminal neuropathic or trigeminal deafferentation pain, depending on the etiology of underlying trigeminal injury.

Pain in the distribution of one or more branches of the trigeminal nerve in association with herpetic eruption represents a separate subtype of facial pain. The acute herpetic pain is usually a self-limiting condition that starts with or just before the development of vesicular rash. Although quite severe, acute herpetic pain responds to narcotic medications and in most cases disappears without any long-term consequences; however, in some cases, especially in older patients, the acute pain transforms into a severe chronic pain condition called *postherpetic neuralgia*.^{22,23}

Most commonly, postherpetic neuralgia involves the territory of the ophthalmic division of the trigeminal nerve. The pain has a burning and dysesthetic character, it is always associated with sensory loss, and allodynia may be a prominent feature. Treatment of postherpetic neuralgia is limited to central neurodestructive

tive procedures, although stimulation of the thalamus and the motor cortex eventually may become an accepted way to manage it.^{24,25} The peripheral nerve stimulation approach has also been tried for postherpetic neuralgia, but the results have been rather underwhelming.¹⁸

The trigeminal nerve is not the only nerve providing sensory supply to the face. Some parts of the face, especially around the ear, are supplied by the nervus intermedius, glossopharyngeal nerve, vagus nerve, occipital nerve, and autonomic afferent fibers that travel through the sphenopalatine ganglion or along the carotid artery. Disturbance of any of these nerves may produce pain in the face. The pain syndromes in these cases are named by their respective suspected pathologic substrates.

Geniculate (nervus intermedius) neuralgia presents with sharp pain deep in the external auditory canal and behind the ear, and may be associated with excessive salivation, tinnitus, and a bitter taste. The underlying mechanism of this pain syndrome is thought to be a vascular compression of the nervus intermedius, and so treatment usually consists of its surgical transection.

Sharp, shooting pain in the posterior part of the tongue, pharynx, tonsils, and ear, sometimes with trigger zones located in the ipsilateral half of the tongue and throat, is characteristic of the *glossopharyngeal* (or *vagoglossopharyngeal*) *neuralgia*.^{26,27} In these cases, the pain may be provoked by swallowing, chewing, and talking. The glossopharyngeal and vagus nerves may be compressed by adjacent vascular structures, so the surgical treatment of this neuralgia resembles that of the typical trigeminal neuralgia and consists of either microvascular decompression of the affected nerve roots or, more commonly, section of the glossopharyngeal nerve and the superior fibers of the vagus nerve.

Pain localized primarily in the occipital area with radiation toward the ear and retromandibular region may represent a more common clinical entity, *occipital neuralgia*. This condition involves the greater or lesser occipital nerves that arise from the second and third cervical nerves, respectively, and somewhat resembles the peripheral neuropathic pain syndrome.^{28,29} The treatment algorithm in this case starts from peripheral nerve procedures with gradual progression to stimulation and ablative procedures, if necessary.^{30,31}

Infraorbital and retro-orbital pain that radiates toward the neck and is associated with lacrimation and conjunctival injection may represent the rare syndrome of *Sluder* (or *sphenopalatine*) *neuralgia*, whereas pain in the frontotemporal region with associated partial Horner syndrome may be caused by an injury to the sympathetic fibers that travel along the wall of the carotid artery (as in cases of carotid dissection) and is called *Raeder* (or *paratrigeminal*) *neuralgia*.³² Other rare pain syndromes include auriculotemporal neuralgia, nasociliary neuralgia, and painful ophthalmoplegia in Tolosa–Hunt syndrome.³³

Vascular headaches and migraines can usually be differentiated from the clinical syndromes mentioned above and are not considered under the rubric of facial pain; however, pain from temporomandibular joint dysfunction and from orofacial pathologic processes should always be a part of differential diagnosis in patients with atypical neuralgias. Neurosurgeons usually see these patients as they are filtered through a series of dentists, primary care physicians, and neurologists, so these patients only rarely reach neurosurgical attention. This, however, may change because peripheral nerve stimulation has now been tried with varying degree of success for both migraine headaches^{34–36} and orofacial pain syndromes.³⁷

The last group of patients does not fit into any of these categories. Their pain commonly does not follow anatomical boundaries, is usually bilateral and diffuse, and is associated with a normal neurological examination, except perhaps for some poorly localized tenderness and vague sensory loss in the painful region. This pain may be classified as *atypical facial pain* and usually represents a form of psychogenic pain disorder. The diagnosis of atypical facial pain may be made if other causes of facial pain have been considered, evaluated, and ruled out; if there is no subjective evidence for most facial pain syndromes; and if specific antecedent or ongoing psychological and behavioral factors can be identified. Obviously, this pain syndrome does not have a surgical treatment. The neurosurgeon's involvement in the management of these cases should end after the diagnosis is established.^{1,2}

■ Nonsurgical Options

Nonsurgical treatment usually is offered to the patient first. In fact, most of these patients see a neurosurgeon for the first time when the conservative treatment fails.

Appropriate pharmacological treatment of facial pain depends on the nature of the pain. The sharp, shooting, electrical pains of trigeminal neuralgia usually disappear completely with oral carbamazepine or oxcarbazepine.^{38,39} This anticonvulsant works so predictably well that pain relief after using it is considered one of the diagnostic criteria for typical trigeminal neuralgia. Over time, however, most patients may need to increase the dose of medication, or the medication may become increasingly less effective, causing the patient to be referred to a neurosurgeon. If, however, the patient develops adverse effects from carbamazepine/oxcarbazepine use, or becomes intolerant of the drug, it may be substituted or supplemented by other anticonvulsants, such as phenytoin, sodium valproate, and gabapentin; antispasticity agents (baclofen); benzodiazepines (clonazepam); or antidepressants (amitriptyline). None of these agents has the same high degree of success as oral carbamazepine or oxcarbazepine, but in some patients, pain may be significantly relieved.

Despite the high level of effectiveness of anticonvulsants, a prospective long-term cohort study of trigeminal neuralgia patients suggested that this effectiveness was rather short lasting, necessitating surgical intervention.⁴⁰ Based on this, the authors concluded that patients may benefit from having surgery earlier in the disease process to improve quality of life, freedom from medications, and the need for regular follow-up.⁴⁰

Opioids may be used for short-term treatment, especially in critical circumstances, when facial pain becomes exacerbated and the definitive pain-relieving procedure cannot be performed immediately. Unfortunately, trigeminal neuralgia is classically resistant to opioids. Gabapentin also has been recommended for a variety of neuropathic conditions, and it may suppress the burning and shooting component of pain in patients with atypical neuralgias and posttraumatic trigeminal pain.⁴¹

Topical ophthalmic application of local anesthetic has been suggested for temporary treatment of pain in first-division (ophthalmic) trigeminal neuralgia; however, a recent double-blind study showed no benefit of administering proparacaine over administering placebo solution.⁴² Topical application of a cream

containing clonidine, an α_2 -adrenergic agonist, in patients with different types of facial pain showed a better effect in neuralgic than in neuropathic pain,⁴³ but this agent is just entering the field of facial pain treatment and needs to be tested more thoroughly.

Nonpharmacological modalities include acupuncture, transcutaneous electrical nerve stimulation, and mechanical vibration. All these treatments were reported to decrease the level of facial pain in some patients, but they do not provide a long-lasting, definitive solution to the problem.^{44–46}

■ Surgical Options

The goal of the surgical treatment of facial pain is to eliminate the pain with minimal morbidity and minimal, if any, new neurologic deficit. This goal can be accomplished by carefully reviewing the surgical indications in each case and by taking into consideration the patient's age and medical condition, presenting symptoms, underlying primary process, personal experience of the surgeon, and the patient's preferences.

Obviously, in cases of secondary trigeminal neuralgia, surgical attention should be directed toward elimination of the offending pathology. Resection of tumors and vascular malformations or systemic treatment of connective tissue disorders and infections may eliminate the pain and sometimes can reverse some of the presenting neurological deficits.

In cases of idiopathic typical trigeminal neuralgia, as well as in some cases of idiopathic atypical trigeminal neuralgia, the underlying pathology is thought to be neurovascular compression of the trigeminal nerve root. Specifically, for typical symptoms to develop, the compression apparently needs to involve the site of transition from peripheral myelin to central, known as *Obersteiner-Redlich zone*,⁴⁷ which is located in the retrogasserian root not far from its entry into the brainstem. Therefore, elimination of this vascular compression should bring complete relief. This theory has proven true with many thousands of patients, and the technique of microvascular decompression of the trigeminal nerve is widely accepted as the most definitive, and the longest lasting, surgical treatment for typical trigeminal neuralgia. It has a high success rate and usually does not cause a significant new sensory deficit in the trigeminal distribution.

This procedure, however, requires retromastoid craniotomy and general anesthesia, making microvascular decompression less desirable for patients who are in poor medical condition, who have short life expectancy, or who do not wish to undergo a major operation. For these patients, the most appropriate means of surgical treatment is a percutaneous procedure that can be done without the prolonged use of general anesthesia. Although neurovascular compression cannot be eliminated through this route, the neuralgic pain may be reliably relieved for several years with minimal morbidity, even though this relief may come with some degree of numbness in part of the trigeminal territory.

Peripheral neurectomies, chemical and thermal neurotomies, and nerve avulsions/exéresis are rarely used for trigeminal neuralgia these days.⁴⁸ Instead, attention is shifted to the gasserian ganglion and retrogasserian trigeminal root, which can be reached percutaneously through the foramen ovale. The three most accepted techniques of this approach are percutaneous radiofrequency trigem-

inal gangliolysis, percutaneous retrogasserian glycerol rhizotomy, and percutaneous balloon compression. All these procedures have their own advantages and disadvantages, but based on a meta-analysis of published studies, it is obvious that each of them is effective in relief of trigeminal neuralgia pain (**Table 10.1**).⁴⁹

The percutaneous procedures are probably the most appropriate means of treatment of trigeminal neuralgia resulting from multiple sclerosis, in which neurovascular compression typically is not considered an issue. These procedures also may work for patients with trigeminal neuralgia secondary to the pontine infarction that involves the root entry zone of the trigeminal nerve.^{14,16}

The next set of procedures involves transection of the trigeminal pathways proximal to the ganglion. This includes open trigeminal rhizotomy, trigeminal tractotomy/nucleotomy, mesencephalotomy, and, most centrally, thalamotomy. Other than rhizotomy, these ablative procedures are used infrequently. Open partial rhizotomy is used in cases of typical trigeminal neuralgia when posterior fossa exploration does not reveal convincing vascular compression of the trigeminal nerve root, or occasionally in patients with multiple sclerosis in whom repeated peripheral ablation has become ineffective. Other central neuroablative procedures are reserved for either atypical trigeminal neuralgia that is refractory to the medical treatment and does not respond to peripheral destructive procedures, for trigeminal neuropathic pain, for anesthesia dolorosa in patients after rhizotomy, or for postherpetic neuralgia.

The last group of procedures includes various neuromodulation techniques, which can be considered in patients with facial pain other than trigeminal neuralgia, such as posttraumatic pain and atypical trigeminal neuralgia. Electrical stimulation, the most common means of neuroaugmentation, can be applied to the peripheral segments of the trigeminal nerve (e.g., the supraorbital nerve), the trigeminal ganglion, the sensory region of the thalamus (ventroposteromedial/ventroposterolateral), or the motor cortex. These procedures have been reported as having varying degrees of success in anecdotal reports or small series of patients

Table 10.1 Results and complications of percutaneous procedures on gasserian ganglion and retrogasserian trigeminal root

Procedure	Radiofrequency thermocoagulation	Glycerol rhizotomy	Percutaneous balloon compression
Number of series	12	13	9
Number of patients	6235	1253	616
Immediate success rate	97%	91.5%	92.3%
Follow-up	6.2 y	2.7 y	2.5 y
Recurrence rate	23.2%	36.2%	17.7%
Anesthesia dolorosa	5.3%	2.6%	0.1%
Corneal anesthesia/ keratitis	12.4%	7.6%	0.1%
Dysesthesia	18%	10.8%	8.4%

Source: Data from Tekkok and Brown.⁴⁹

toward less invasive nonablative procedures, a better understanding of pain mechanisms, and optimization of pharmacological therapy will continue to improve the neurosurgical approach to facial pain treatment in the future.

Editor's Comments

This chapter is a complete description of the diagnosis and surgical management of trigeminal neuralgia. Many of these "surgical options" are discussed in upcoming chapters. As we point out in this survey, the evidence to support surgical decision making in trigeminal neuralgia is somewhat lacking. We know what works; we know generally how long it works. We have a sense of which procedures might be appropriate, given the nature of pain, and the age and health of the patient. The algorithm suggested is merely a way to think about surgical care. Fortunately, we do have a number of surgical options for patients with trigeminal neuralgia who develop medical intractability.

This chapter points out as much about what we don't know about trigeminal neuralgia as it does about our current understanding of this disorder. We have by no means a complete and rigorous understanding of facial pain. I anticipate that progress in this area will continue, and much more is still to be recorded on this topic.

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Microvascular Decompression for Trigeminal Neuralgia

Kim J. Burchiel

In 1934 Dandy¹ first proposed that vascular compression of the trigeminal nerve was a cause of trigeminal neuralgia (TN). However, Dandy also pointed out that vascular contact occasionally occurs without the production of TN, and that TN may be present in the absence of trigeminal nerve compression. Contact of the trigeminal nerve by a blood vessel at or near the root entry zone^{1–3} is still thought to be the most common anatomical association in patients with TN. Devor's "ignition theory" of TN relies on a focus of hyperactivity in the retrogasserian root, consistent with a focus of demyelination within the nerve, possibly caused by vascular compression. The theory holds that "nerve injury results in hyperexcitability of injured afferents, which results in after-discharges large enough to result in nonnociceptive signal being perceived as pain. This leads to windup and both peripheral and central sensitization."⁴ Other etiologies such as multiple sclerosis,⁵ central or peripheral demyelination,^{6,7} root injury, and tumors may be associated with TN.^{2,8}

It was Gardner who first suggested that vascular decompression of the trigeminal nerve could ameliorate TN.^{9,10} Subsequently, Jannetta's extensive work established microvascular decompression (MVD) as one of the most important surgical approaches to TN and other cranial neuropathies.^{3,11} Over more than four decades, MVD has established itself as a unique and durable treatment for medically intractable TN, a procedure that seemingly has dissociated the need for nerve injury, the attendant sensory loss, and a long-term successful outcome.

Vascular compression of the trigeminal nerve is also known to occur in patients who do not have TN, and TN is known to occur in patients who have no vascular compression. In 1982 Adams et al reported on "our failure to be convinced by vascular compression as a cause for the majority of our patients' pain."¹² He questioned the role of "microvascular compression" in the surgical treatment of TN, reviewing all the evidence at the time. He made the argument that there may be some other process involved.¹³ In contrast, Jannetta and others have reported that essentially all patients with TN are found to have vascular contact with the nerve, at times from both arteries and veins.^{14,15}

A review of autopsy studies reveals some degree of contact between the trigeminal nerve and a blood vessel in 90 to 100% of patients with TN, but also in 16 to 58% of patients without TN.^{16–19} In 2009 Miller et al²⁰ evaluated neurovascular compression (NVC) in patients with and without TN and concluded that trigeminal NVC occurred in 17% of asymptomatic patients but was more severe and more proximal in patients with TN. A review of the literature reveals a wide range (4 to 89%) of TN patients with no demonstrable vascular contact.^{1,12,19,21–25} For example, Leal et al²⁴ reported no NVC in 9% of patients by surgical exploration and imaging revealed no vessel in relation to the nerve in 12% of patients. Improve-

ments in imaging technology resolution capabilities have further reinforced the proposition that TN can occur in the absence of vascular contact.^{24–26}

Hamlyn in 1992 noted that an explanation for TN cases in which no vessel was found in contact with the trigeminal nerve at operation was needed, and that it should be possible to identify those cases preoperatively.¹⁹ In 2009 Miller et al. stated that “trigeminal NVC occurs in asymptomatic patients but is more severe and more proximal in patients with TN.”²⁷ Although vascular compression of the trigeminal nerve by a blood vessel at or near the root entry zone^{1–3} is the primary pathology of TN, there remains an unexplained subset of patients with TN without clear NVC. Improvements in surgical approaches and advances in imaging technology have only reinforced this discrepancy.^{8,12,18,19,21,22,28}

A retrospective review of patients with TN type 1 (TN1) or type 2 (TN2)²⁹ at Oregon Health & Science University from July 2006 to February 2013 was recently undertaken.³⁰ Patients underwent preoperative high-resolution magnetic resonance (MR) imaging and analyzed MR angiograms with 3D reconstructions using OsiriX. MR imaging (BFFE and MRA) using a 3-T MR scanner was as previously described.^{25,27}

Our review yielded 257 patients with TN1 (219 patients) and TN2 (38 patients) who underwent high-resolution MRI/MRA with 3D reconstruction of combined images. Of the 257 patients, 33 had undergone previous MVD procedures, 26 TN1 patients and 7 TN2 patients. Of the 219 TN1 patients, 150 (68.49%) demonstrated unilateral compression, 6 (2.74%) had bilateral compression, and 63 (28.8%) had no NVC. The identity of the offending vessel was the superior cerebellar artery (SCA) in 121 cases (74.7%), venous in 20 (12.4%), anterior inferior cerebellar artery (AICA) in 13 (8%), vertebral artery in 5 (3.1%), basilar artery in 2 (1.2%), and a tumor in 1 (0.62%, meningioma).

Six TN1 patients had bilateral TN1 symptoms at some point in their history. Of these, 2 patients had bilateral compression, 1 patient had unilateral compression, and 3 patients had no compression as determined by imaging. Therefore, in patients with bilateral TN1, only 5 of 12 (41.67%) had visible NVC on imaging studies. Of the 38 TN2 patients, 31 (81.58%) had unilateral compression, and the vessel was the SCA in 26 (68.42%), a vein in 3 (7.89%), and AICA in 2 (5.26%). There were no patients with bilateral NVC and 7 had no NVC (18.4%).

Of the four TN2 patients with bilateral TN2 symptoms, no patient had bilateral compression, 3 patients had unilateral compression, and 1 patient had no compression. Therefore, in this group only 3 of 8 (37.5%) had visible compression on imaging studies. Sensitivity of imaging as a predictor of NVC for both TN1 and TN2 was 96%, and specificity of imaging findings for patients with TN1 and TN2 were 90% and 66%, respectively.

Thus, it appears that TN1 and TN2 can occur without NVC, and that preoperative imaging can, in most cases, determine if NVC is present.

■ Indications

When the diagnosis of TN is recognized, medical therapy should be attempted in essentially every patient. Occasionally, patients are so intolerant of anticonvulsant medications, due to either side effects or allergic reactions, that surgical therapy is considered at the outset of the disorder. More commonly, patients can

be adequately treated with medications for several years, or even longer, given the sporadic nature of the disorder early on. Over time, TN becomes more persistent, and also becomes more medically resistant. At this point the diminished efficacy of medical management and emergent toxicity of anticonvulsant therapy can result in the clinical conclusion that the patient is approaching medical intractability.

The indications for an MVD for TN are: (1) medical intractability of the pain, (2) medical fitness for a posterior fossa craniotomy, (3) the presence of NVC demonstrated by imaging, and (4) patient choice of the procedure. In comparison to destructive procedures, MVD has the longest duration of pain relief,^{31,32} and for this reason it is generally considered in younger patients, those with the greatest longevity. In the past, patients over age 70 have not been considered optimal candidates for the surgery, although this concept has been challenged by the demonstration of the relative safety of this procedure in an older patient cohort.³³ Clearly, destructive procedures can generally and adequately control TN in older patients over their life spans, given their reduced longevity. However, it is also clear that when older patients are subjected to destructive procedures they are more likely to develop complications of deafferentation, such as anesthesia dolorosa and its variants, in comparison with a younger population. These two facts may warrant a reconsideration of MVD for older patients with TN.

■ Techniques

MVD is performed under general endotracheal anesthesia, using a minimum of inhalational anesthetic and no chemoparalysis. A bipolar facial electromyogram (EMG) is recorded ipsilaterally from the orbicularis oris and orbicularis oculi. Brainstem auditory evoked responses (BAERs) are recorded bilaterally.

The patient is placed supine, with the head placed in the Mayfield head holder. The head is rotated away from the side of surgery, and the neck is slightly flexed and elevated (**Fig. 11.1**). **Fig. 11.2** shows the anatomical relationships of the retromastoid approach. After sterile preparation of the skin, a curved retroauricular incision is made from approximately the top of the pinna to the mastoid notch (**Fig. 11.3**), and the skin edges are retracted using “fish hooks.”

A small craniectomy is then created by drilling, initially taking care to stay inferior and posterior to the asterion. The craniectomy is then enlarged with Kerrison rongeurs to the point that the edges of the anterior transverse sinus, transverse-sigmoid junction, and the superior aspect of the sigmoid sinus are minimally, but definitively, exposed. Any bleeding points are controlled with small pieces of Gelfoam (Pfizer, New York, NY, USA) soaked in thrombin. The final size of the craniectomy should be 2.5 to 3.0 cm at its base and 3.0 to 3.5 cm at its surface to create a beveled edge (**Fig. 11.4**). Opened mastoid air cells should be fully waxed.

The dura is then opened in a curvilinear fashion paralleling the transverse-sigmoid junction, and the dura anterior to the incision is tacked up using three or four sutures. One or two small cottonoids are then inserted through the incision and over the cerebellum to facilitate cerebrospinal fluid (CSF) drainage. A flexible retractor is used to hold a small rounded brain retractor blade, and this is inserted to exert gentle pressure on the cerebellum and to augment CSF drainage. At this point the operating microscope is brought into position.

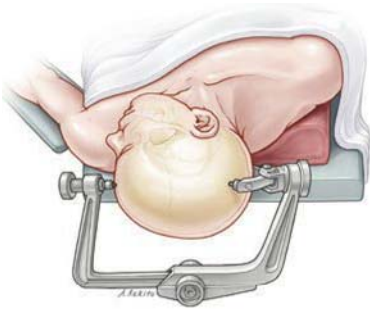


Fig. 11.1 Mayfield positioning.



Fig. 11.2 View of the retromastoid region showing the relationships of the top of the pinna to the transverse sinus, the position of the transverse-sigmoid junction and sigmoid sinus to the asterion, and the relationship of the mastoid to the sigmoid sinus.



Fig. 11.3 Retroauricular skin incision, and relationship to planned craniectomy.

Under magnification, and under direct vision, the retractor blade is then inserted over the cerebellum pointing directly at the interval between the tentorium and the petrous ridge, taking care to not inadvertently injure any branches of the petrosal vein, which can be multiple and can be distributed anywhere along the tentorial-petrous junction (**Fig. 11.5a**). Most commonly, when the petrous vein is encountered, an apparent trifurcation points to a common entry into the superior petrosal sinus. The entirety of the petrous

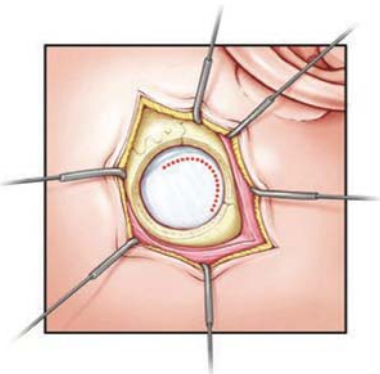


Fig. 11.4 The skin and underlying musculature are retracted using "fish hooks," and the 2.5- to 3.5-cm craniectomy is made to just expose the inferior margin of the transverse sinus, the transverse-sigmoid junction, and the posterior extent of the sigmoid sinus. A curvilinear durotomy is made to 3–4 mm posterior to the transverse-sigmoid junction, and its anterior margin is retracted using three or four dural sutures.

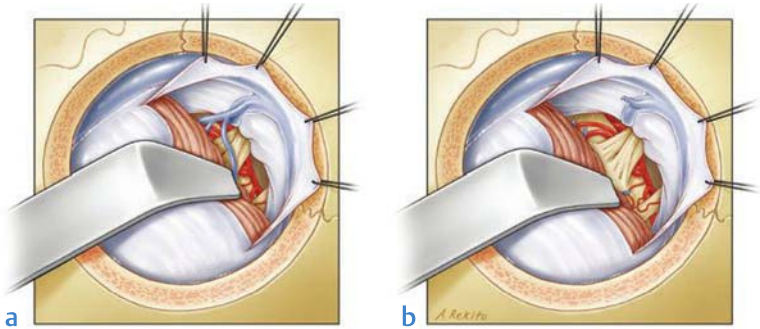


Fig. 11.5 (a) Retraction is used to facilitate CSF (cerebrospinal fluid) drainage and to expose the tentorial-petrosal angle with the petrosal vein(s). (b) The petrosal veins are typically coagulated and divided to facilitate exposure and retraction.

vein is typically coagulated by bipolar cautery, with careful irrigation, and divided (**Fig. 11.5b**). The retractor is then inserted deeper over the cerebellum to eventually uncover the vestibulocochlear and facial nerves. More superiorly and at the apex of the exposure lies the trigeminal nerve, and its entry into the brainstem.

Once the trigeminal nerve is located and exposed, dissection of the offending vessel is begun. This is typically the superior cerebellar artery (SCA), and this vessel can usually be liberated from beneath the trigeminal nerve superiorly (**Fig. 11.6a, b**). The rare compression from the AICA is resolved inferior to the nerve. If the vertebral or basilar artery is the source of compression, mobilization of the artery is usually not completely successful.

During the procedure careful attention is paid to the ipsilateral BAERs. A latency change of less than 10% in waves IV and V is not unusual, and is almost

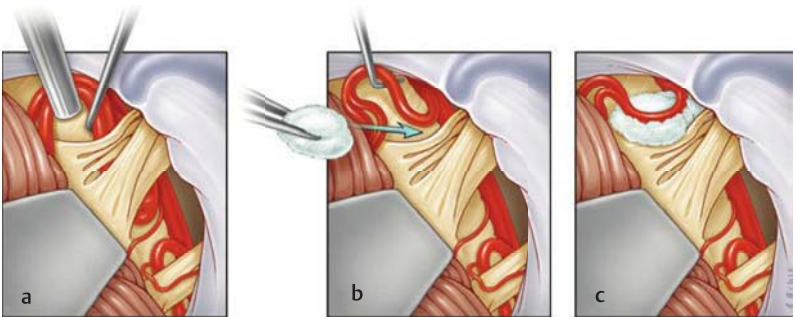


Fig. 11.6 (a) Vascular compression of the nerve is demonstrated, which in most cases is due to the superior cerebellar artery (SCA). (b) The artery is mobilized over the lateral surface of the nerve. (c) Teflon fiber packing is used to maintain the position of the artery away from the nerve.

immediately reversible at the conclusion of the procedure. If the BAER latency is delayed beyond 10%, or if amplitude changes in the BAERs are observed, the retractor is removed, and usually the decompression can proceed without additional retraction. If not, the retractor can be replaced once the amplitude changes have resolved, and BAERs can again be monitored for recurrent amplitude change.

Small pieces of Teflon (DuPont, Wilmington, DE, USA) fiber pledget, teased into small loose balls, are then placed in the interval between the nerve and artery, so as to securely and permanently separate the two structures (**Fig. 11.6c**). For highly redundant arteries, care must be taken to avoid kinking the artery during the placement of the Teflon balls. In the case of vertebral or basilar compression, usually the most that can be accomplished is the separation of the nerve and artery, although packing Teflon fibers between the nerve and these large arteries places the nerve under considerable tension.

Once the decompression is accomplished, hemostasis is ensured, and all retractors are removed. The dura is closed and sealed with fibrin glue, and the small skull defect is repaired with hydroxyapatite cement. The muscle and skin are closed in layers.

■ Outcomes

Despite the seeming complexity of MVD surgery, reported complication rates are low. CSF leakage can occur in 1.5% of patients,³¹ hearing loss in 0.59%,¹⁴ meningitis in 0.37%, cerebellar or supratentorial hematoma in 0.3%, permanent facial paresis in 0.15%, permanent extraocular palsy in 0.15%, and operative death in 0.15%.³¹ With no complications, patients are usually able to return to full activities and employment within a month, although lingering effects of the surgery may last for several months.

Trigeminal neuralgia recurrence rates after an initially successful MVD have been reported from 1.7 to 75% (**Table 11.1**).^{28,31,32,34–50} Van Loveren et al reported that of 50 TN patients treated by MVD 84% were pain free at 3 years.⁴⁸ Liao et al. found that of 80 MVDs there were 5 cases of recurrence in 12 months.⁴¹ Barker et al examined 1,185 patients who underwent an MVD for TN over a 20-year period. Patients were followed for 1 year or longer after surgery, with 91% having follow-up of at least 5 years. The SCA was the cause of compression in 75% of patients, and the AICA in 10%. A compressive vein was observed in 68% of patients, and in 12% a vein was the sole cause of compression. Recurrence of TN was found in 282 (23.8%) patients and reoperation was performed in 132 patients. They observed higher rates of recurrence: (1) in women, (2) in patients with 8 years of symptoms prior to surgery, (3) with decompression of a vein during surgery, and (4) in association with a lack of immediate postoperative relief.³¹

Bederson and Wilson reviewed 252 MVDs performed in 246 patients. Thirty patients (12.2%) had no observable compression and received a partial sensory rhizotomy (PSR), and 56 patients had vascular contact without distortion and received an MVD with a PSR.³⁴ Zakrzewska and Thomas evaluated 475 patients with TN. Sixty-five MVD procedures were performed in 55 patients; at 5 years 38% of the MVD patients had experienced a TN recurrence.⁴⁹ Rath et al reported on 135 MVDs with a follow-up interval of 3 months to 5 years (mean 1.4 years). Venous compression was seen in 9 patients and 7 patients had no venous or arterial com-

Table 11.1 Summary: Lack of compression and recurrence rates for microvascular compression and radiofrequency lesioning procedures

Series	MVD (N)	RFL (N)	Follow-up	No compression (%)	MVD recurrence rate (%)	RFL recurrence rate (%)
Burchiel 2013	184		Mean 44.9 mo	16	22	
Van Loveran et al 1982	50	700	3–6 y		12 (at 3 y)	20 (at 6 y)
Liao et al 1997	80		9 mo–4 y		6.25 (at 1 y)	
Barker et al 1996	1,185		> 1 y		23.8	
Lee et al 2000	393		≤ 5 y		40.6 (≤ 3 mo); 15.6 (≤ 6 mo); 18.8 (≤ 12 mo); 12.5 (≤ 3 y); 3 (≤ 5 y)	
Zorman (Wilson) 1984	125		6 mo–13 y	20.8	16 (2.4 y)	
Bederson (Wilson) 1989	246		5.1 y	12.2	8.13	
Taha (Tew) 1997		1,200	7–9 y			20 (7–9 y); 25 (15 y)
Zakrzewska (Thomas) 1993	55	265	Mean 45 mo		13 (mean 45 mo)	29 (mean 45 mo)
Rath et al. 1996	135		3 mo–5 y; mean 1.4 y	5	16.7 (5 y)	
Sun et al. 1994	61		1.1–10.5 y		16 (≤ 2 y)	
Burchiel et al. 1988	36	8*	Mean 8.5 y		47	50
Tronnier et al. 2001	225	206	Mean 10.9 and 14.0 y		23.6 (2 y); 35 (10 y); 37 (20 y)	50 (2 y); 75 (4.5 y)
Kanpolat et al. 2001		1,600	Mean 68.1 mo			7.7 (< 6 mo); 17.4 (> 6 mo)
Sindou et al. 1990	120		41 mo			
Klun 1992	178	42*	12 y; mean 5.2 y		6	50
Cutbush (Atkinson) 1994	109		Mean 4.8 y		24	
Mendoza (Illingworth) 1995	133		6 mo–15 y; mean 5.3 y		16	
Kondo 1997	281		5–20 y; mean 10.1 y		16.7	
Lee et al. 1997	146	235	Mean 5.7 y	2 pts	8.6	45.7 (glycerol)
Chen (Lee) 2003	114	127	≤ 2 y	4 pts	17.5	8.7

Abbreviations: mo, month; MVD, microvascular decompression; N, number; pts, patients; RFL, radio-frequency lesioning; y, year.

*Partial sensory rhizotomy.

pression (5%). Their recurrence rate was 16.7% at 5 years. They also commented that patients who had previous destructive procedures had worse outcomes.⁴³

Sun et al reviewed 61 patients after MVD for TN with follow-up of 1.1 to 10.5 years (mean 6.7). There were 10 (16%) patients with recurrence within 2 years postoperatively.⁴⁵ Tronnier et al reviewed 225 MVDs and found that 63% of patients had 20 years of pain relief.⁴⁷ Sindou et al evaluated 120 patients after MVD, with a follow-up period of 41 months. They reported that 83.3% and 79% of patients had pain relief from sitting craniotomy and the lateral approach, respectively.⁴⁴ Cutbush and Atkinson evaluated 109 MVDs from a single surgeon. Their mean follow-up was 4.8 years and 83 (76%) patients had resolution of their pain. They commented that most (66%) of the recurrences occurred within 12 months and the SCA was the vessel involved in over 70% of the cases.²⁸ Mendoza and Illingworth evaluated 132 patients who had 133 MVDs (1 patient with bilateral symptoms). Follow-up in this series was from 6 months to 15 years with an average of 5.3 years. There were 95 (71%) patients who were pain free and 21 (16%) patients with a minor recurrence. Operative findings of compression were statistically significant in giving long-term pain relief.⁴² Lee et al evaluated 116 patients after MVD followed up 2 years postoperatively and there were 9 (8.6%) recurrences. There were at least 2 patients with no vascular compression who had pain recurrence.³⁹ Chen and Lee reviewed 114 MVDs with a follow-up of at least 2 years, with a recurrence rate of 17.5%. They noted that 4 patients (3.5%) did not have any compression visible at the time of surgery, and 9 patients had a prominent bone spur or acute angulation at the entry into Meckel cave as the proposed cause of compression.³⁵

Burchiel et al³² reported long-term results after MVD for TN, with an average follow-up of 8.5 years. In their series, during the first 2 years there was an approximately 25% recurrence rate, after which results remained stable until 5 years postoperatively, at which point recurrences began to mount. Over the course of follow-up pain recurrences averaged approximately 4% per year, and showed no trend toward plateauing (**Fig. 11.7**). In a later report, Miller et al²⁰ conclusively demonstrated what was widely believed to that point, that after MVD the outcome of TN1 was superior to that for TN2 (**Fig. 11.8**).

Long-term studies on the outcome from rhizotomy for TN are not as robust as those for MVD. Burchiel et al showed that after MVD or trigeminal rhizotomy (bipolar cautery of the lateral two thirds of the trigeminal nerve after crushing) recurrence occurred in 47% of the MVD group and in 50% in the trigeminal rhizotomy group, with a mean follow-up period of 8.5 years.³² Zorman and Wilson evaluated 125 MVDs and PSRs. The mean follow-up was 26 months (6 months to 13 years). In 26 patients there was no compression seen at the root entry zone and each of those patients underwent a PSR. They reported a 91% success rate for pain relief.⁵⁰ Klun evaluated 178 patients after MVD and 42 patients after PSR with a follow-up period of up to 12 years (mean of 5.2 years). Eleven patients had tumors, 3 patients had MS, 4 patients had bilateral pain, and he excluded patients with "atypical pain." Five-year complete relief was 84% for both operations.³⁷

It is important to note in most of the series mentioned above there was no differentiation of TN1 and TN2 symptomology, no confirmatory preoperative imaging, and no indication of the degree and severity of NVC.

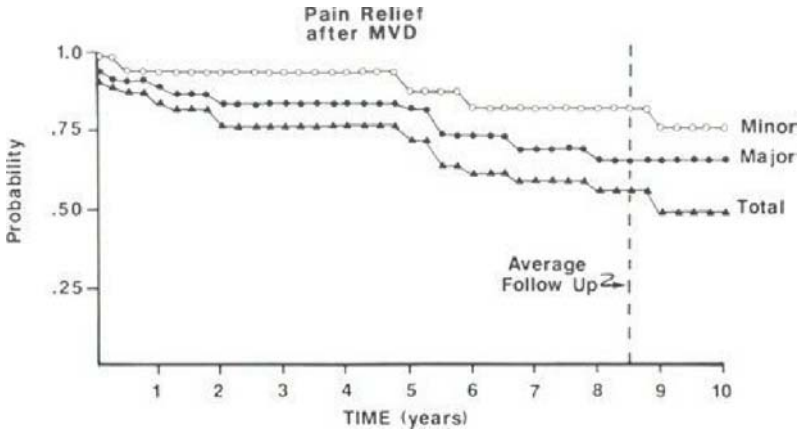


Fig. 11.7 Kaplan-Meier plot of probability of pain control over time after MVD (microvascular decompression). Major recurrences required additional surgical therapy, and minor recurrences required only medication for pain control. The total recurrence rate is approximately 4% per year.

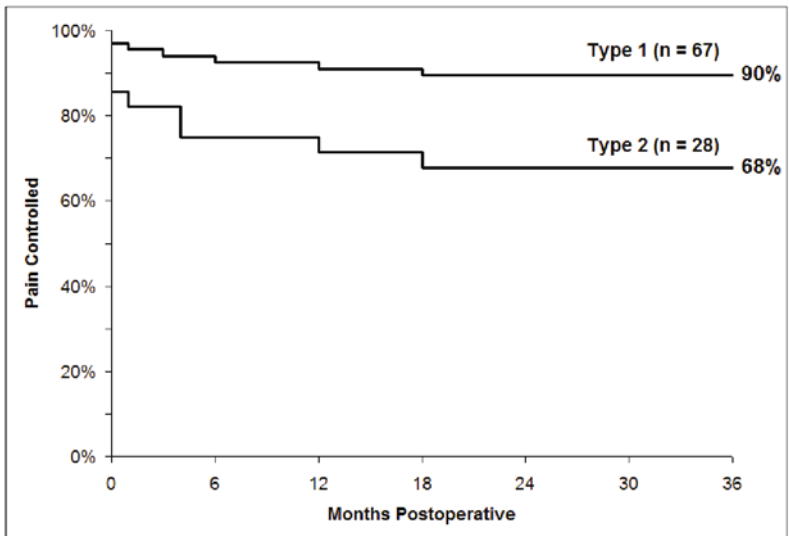


Fig. 11.8 Kaplan-Meier plot of pain control over time after MVD (microvascular decompression) in TN (trigeminal neuralgia) 1 and TN2.

From the studies cited above, two broad conclusions can be drawn. First, there seems to be a general consensus that some patients with TN do not manifest NVC at the time of surgical exploration. The mechanism for TN in these cases is unexplained. Second, despite initially effective MVD, the rate of pain recurrence may be as high as 4% or 5% per year.^{1,12,19,21–25} There is no consensus as to the etiology of pain recurrence in these cases.

TN patients often experience a recurrence after an initially successful MVD procedure. A review of the literature reveals a wide recurrence range, 7.7 to 75%.^{28,31,32,34–50} Surgical alternatives after recurrence include repeat exploration for recurrent vascular compression (MVD), internal neurolysis (IN), and radiofrequency lesioning (RFL). Alternatives for recurrent TN include partial or complete sensory rhizotomy, balloon rhizotomy, glycerol injections, and radiosurgery. Most experienced surgeons tend to favor less destructive approaches because anesthesia dolorosa (deafferentation pain) is a known, and feared, complication of destructive procedures of the trigeminal nerve.

Our results suggest that high-resolution MRI/MRA imaging can reliably detect NVC in patients with TN. Our imaging indicates that 28.8% of patients with TN1 *do not* manifest NVC. Further, post-MVD imaging can discern whether trigeminal NVC has been effectively relieved. A result of these postoperative studies is the recognition that *most* patients who exhibit clear imaging evidence of prior MVD of the nerve have recurrent TN *without* recurrent NVC. The main exception to this appears to be when the original NVC was either missed or inadequately decompressed at the time of the first MVD.

We have previously demonstrated that 17% of the general population manifests NVC of the trigeminal nerve.²⁷ If, as has been estimated, the incidence of trigeminal neuralgia in the population is 1 in 10,000 (0.01%),⁵¹ then 99.94% of individuals with trigeminal NVC *do not* have TN. Given these statistics, and the present evidence that TN can both occur and recur without NVC, the hypothesis that TN is reliably caused by neurovascular conflict must be challenged.

There is no serious debate that MVD is the most efficacious procedure for medically intractable TN. The question is why MVD works at all, when neurovascular compression may just be one element—and not an essential one—in the genesis of TN. Rather than “decompression,” MVD may be effective because it creates chronic stretching and compression of the nerve, or it may be that in some way the arachnoiditis and scarring that accompany MVD either change the blood supply or otherwise alter the physiology of the nerve. These and other questions, including the possibility that genomic predisposition to the development of TN may play an important role, need to be addressed by clinical and laboratory research.

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Trigeminal Rhizotomy

Shih-Shan Lang and John Y. K. Lee

There are numerous surgical procedures used for the treatment of trigeminal neuralgia (TN), of which microvascular decompression (MVD) is the most common. This popular procedure is performed at the root of the trigeminal nerve (defined as the section of nerve between the gasserian ganglion and the brainstem). However, in a small population of patients, vascular compression is not seen at the time of surgical exploration.¹⁻⁸ These patients may benefit from an alternative procedure, partial sensory rhizotomy (PSR). The level of evidence for these procedures remains poor: All studies are Class III (retrospective case series or expert opinion only). Therefore, readers should recognize that the quality of the evidence for performing PSR is low.

■ History of Trigeminal Rhizotomy

Prior to the development of rhizotomy, multiple peripheral neurectomy procedures were performed in an effort to cure trigeminal neuralgia. Targeting the infraorbital sensory nerve or the mandibular nerve, for example, resulted in only temporary relief. Thus, surgeons explored more invasive alternatives to cure patients with intractable trigeminal neuralgia. In 1891 Sir Victor Horsley was the first to describe trigeminal rhizotomy as the sectioning of the trigeminal root between the brainstem and the gasserian ganglion.⁹ He reported that complete removal of the gasserian ganglion was not possible without devastating outcomes. Therefore, he performed a trigeminal root sectioning (rhizotomy) in a patient suffering from TN through a subtemporal craniotomy. Throughout the next decade, several surgeons described the suboptimal results from gasserian ganglionectomy and proposed subtotal trigeminal neurectomy in order to preserve the motor root.¹⁰⁻¹² For example, Hartley and Krause independently modified the technique from an intradural operation to an extradural operation.^{13,14} However, it was not until 1901 that the procedure of dorsal sensory root sectioning through a middle fossa approach was popularized by University of Pennsylvania neurologist and neurosurgeon Spiller and Frazier.¹⁵ In 1925 Frazier further refined this procedure by describing a subtotal neurectomy approach that preserved not only the motor root, but also the ophthalmic division fibers that provide corneal sensation.^{16,17} In the same decade, Stookey further advanced this procedure by differentiating the fibers in the dorsal root derived from the mandibular division from those derived from the maxillary division.⁶ Thirty years later Stookey described his experience with over 700 trigeminal rhizotomies and reported 92% pain-free outcomes.¹⁸ However, overall complications were high: 8% of patients had facial weakness, 29% had mild paresthesias, and 10% had severe paresthesias. Also, in the 1950s, Peet and Schneider reported their 10-year experience on 553 patients who un-

derwent trigeminal rhizotomies.⁴ Similar to the Stookey study, a subtemporal craniotomy was performed under local anesthesia and the majority of these operations were performed in the sitting position. Similarly, pain relief was high, at 95%; however, the majority of patients had some type of significant parasthesias, and facial paralysis occurred at a rate of 6.5%.⁴

The subtemporal approach was the most popular craniotomy during this premicroscope era. However, in the 1920s Walter Dandy proposed the retromastoid/posterior fossa approach to the trigeminal nerve. He sectioned the sensory root at the level of the pons.¹⁹ He reported the hypothesis that, due to the position of the pain fibers running in the posterior portion of the trigeminal nerve, he could spare the normal facial sensation via a posterior fossa approach. Dandy reported outcomes in 250 patients with this approach, only 4 of whom had recurrences.²⁰ In his series, there were no cases of keratitis, or facial nerve or motor root injury. In addition, he found 18 cases of posterior fossa tumors that were the cause of the TN, which would not have been found during a subtemporal approach. Despite these successful results, neurosurgeons during his era did not or could not adopt this approach. Walter Dandy is often credited for having incredible skill and keen vision, operating in the posterior fossa without the advantage of microscope or modern illumination. Few surgeons in that era were able to adopt Dandy's technique with the same success rate, and thus the subtemporal route remained the approach of choice.

Over the next few decades, trigeminal rhizotomy via subtemporal or retrosigmoid craniotomy became less popular with the advance of antiepileptic medications as well as the innovation of less invasive, percutaneous techniques described in other chapters. In addition, the arrival of the microscope led to an alternative, nondestructive technique. In 1967 Peter Jannetta confirmed observations of Walter Dandy in the posterior fossa, describing the etiology of trigeminal neuralgia as being vascular compression.¹ With the advantage of the microscope and electrocautery, Jannetta achieved high success rates with vascular decompression using Teflon (Pfizer, New York, NY, USA) implants to displace the artery/vein away from the nerve.¹ With the gradual success of Jannetta's microvascular decompression, craniotomy for trigeminal rhizotomy became virtually a salvage operation.^{1,5,7,21,22} In the current era, trigeminal rhizotomy is typically reserved for the patients who have undergone a MVD with no evidence of a compressing vascular structure or recurrent TN with poor outcomes.

■ Literature Review

We conducted a PubMed search for the following terms: "trigeminal neuralgia, microvascular decompression, partial sensory rhizotomy." We defined the search to include both microvascular decompression (MVD) and partial sensory rhizotomy (PSR) because in current practice, patients are typically offered the Jannetta MVD, and PSR is usually performed only during a negative posterior fossa exploration. Thus, it is unusual to schedule a patient for primary up-front PSR without knowledge of the status of vascular compression or without having performed a microvascular decompression previously. We also excluded studies on patients with multiple sclerosis and studies performed before 1989, which was

the approximate date marking the entrance of magnetic resonance imaging (MRI) scans into routine clinical practice. We identified 33 papers and included 13 in our analysis. All studies were retrospective case series—Class III evidence. This is summarized in **Table 12.1**. The overall rate of performing a PSR during posterior fossa exploration for trigeminal neuralgia was 28% (438 of 1,538 patients). Hence, among surgeons who keep PSR in their armamentarium for surgical procedures, approximately one quarter of the patients will undergo a partial sensory rhizotomy at the time of surgery. This practice obviously excludes surgeons such as Dr. Jannetta himself, who published on 1,185 patients in 1996 and did not report having performed a partial sensory rhizotomy on any of them.²³

Table 12.1 Literature review of studies including microvascular decompression (MVD) and partial sensory rhizotomy (PSR)

	Patients (N)		Pain-free outcome: 1 year (%)		Pain-free outcome: 2 years (%)		Pain-free outcome: 5 years (%)	
	MVD only	MVD + PSR	MVD only	MVD + PSR	MVD only	MVD + PSR	MVD only	MVD + PSR
Zhang et al. 2012 ³⁷	142	68	90.6	92.5	90.2	97.8	–	–
Pollock et al. 2011 ³⁴	59	8	87*	87*	–	–	78*	78*
Ma et al. 2009 ³³	86	10	–	–	–	70 (3 yrs)	–	–
Zakrzewska et al. 2005 ³⁶	245	60	–	–	–	–	79	72
Liu et al. 2004 ³	0	40	–	80	–	–	–	–
Delitala et al. 2001 ²⁵	34	14	–	–	87.5	12.5	–	–
Howng et al. 1998 ²⁶	0	8	–	–	–	–	–	62.5
Walchenbach et al. 1994 ³⁵	51	5	–	–	–	–	71*	71*
Cho et al. 1994 ³¹	376	24	86*	86*	–	–	–	–
Young et al. 1993 ³⁰	152	102	–	58	–	56	–	50
Jamjoom et al. 1992 ³²	49	11	–	–	88*	88*	–	–
Klun et al. 1992 ²⁷	178	42	–	–	–	–	94	51
Bederson et al. 1989 ²⁴	166	86	–	–	–	–	75*	75*

* Equal percentages due to outcomes not distinguished between the two groups.

■ Technique for Partial Sensory Rhizotomy

Because PSR is not typically the first-line surgical treatment, there are limited literature reports describing PSR in the current era.^{3,24–37} In all the literature reports, the choice of performing partial rhizotomy instead of traditional MVD was done on a case-by-case basis depending on whether a significant vascular structure was encountered during posterior fossa exploration. The variability in technique is described as follows. In a large series by Young et al 102 patients underwent a PSR.³⁰ The authors used intraoperative brainstem auditory evoked responses (BAER) and somatosensory evoked potentials (SSEP). In 74 patients who underwent PSR, one third to one half of the cross-sectional area of the sensory root was incised from its caudolateral part approximately 2 to 5 mm from the pons. In the other 9 patients, approximately two thirds of the nerve root was sectioned. No patients underwent a complete neurectomy. Klun reported his experience with partial sensory rhizotomy in 42 patients with a mean follow-up of 5.2 years.²⁷ His surgical technique consisted of sectioning one third or less of the portio major, with consideration of the distribution of trigeminal branches. The rhizotomy was performed as close as possible to the brainstem. Similarly, Zhang et al performed a PSR by cutting one fifth to one quarter of the breadth of the sensory root with scissors as close as possible to the brainstem using no coagulation.³⁷ In both case series by Liu and Apfelbaum³ (40 patients) and Pollock et al³⁴ (8 patients) a partial rhizotomy of the trigeminal nerve consisted of sectioning approximately one third to one half of the nerve next to the brainstem, starting at its posterior inferior margin. They describe first scoring the pia with microscissors and then lengthening this resection using a microhook. They did not alter this technique based upon the locality or division of the patient's TN. Delitala et al reported on 14 patients who underwent a partial sensory rhizotomy; up to half of the inferolateral “portio major” was sectioned.²⁵ In another series of 8 patients, rhizotomy was performed with a microdissector and the extent of rhizotomy was determined by the patient's pain involvement, ranging from approximately one quarter to three quarters of the nerve²⁶ (**Fig. 12.1**).

In summary, the technique of partial sensory rhizotomy varies among different surgeons. Most surgeons perform partial sectioning using microscissors. The anatomical positions of the fibers that innervate each branch of the trigeminal nerve have been studied.^{38,39} The fibers from the mandibular division are found ventral-lateral throughout the length of the ganglion to the pons, the ophthalmic division fibers dorsomedial, and the maxillary division fibers in between. Thus, a partial sensory rhizotomy is usually performed by cutting the ventral (caudal) -lateral portion of the trigeminal nerve, although there is some variability, especially because the nerve (and its somatotopy) may rotate slightly upon exiting the pons and entering Meckel cave. In addition to direct sectioning techniques, Ma et al reported a different technique termed “nerve combing.”³³ This technique resembles neurolysis in that a “micro-needle” is used to separate individual fascicles of the trigeminal nerve. In personal communication with Dr. Michael Lim from Johns Hopkins University and in the senior author's (JYKL) own experience, alternative techniques have included direct injection of glycerol into the cisternal portion of the nerve. The efficacy of these different techniques cannot be compared directly.

■ Results and Outcomes

Overall, mixed results have been reported with PSR. **Table 12.1** shows our analysis of the literature review, depicting a trend toward poorer outcomes in the PSR group. Many series reported combined outcomes for the MVD-only group and the MVD + PSR group because a significant difference between the two groups could not be accounted for.^{24,31,32,34,35} In one of the largest case series, Zakrzewska et al described 245 patients who underwent MVD only and 60 who underwent

PSR.³⁶ Interestingly, the PSR group experienced the lowest satisfaction rate, with 4% of MVD patients reporting themselves as “not satisfied” versus 20% of PSR patients. Complications from PSR were the leading cause of poor outcomes in the PSR group. However, one can also speculate that the patients who required PSR have a different etiology of pain that may lower the potential success of any particular technique.

In the series by Young and Wilkins,³⁰ 83% of their 102 patients were pain free at 1 year follow-up, with no or mild sensory deficits in 82% of patients. The two main variables they reported as predictive of a poor pain outcome included reoperations and lack of preoperative involvement of the mandibular division. Bederson and Wilson performed a PSR on 30 patients in whom no vascular structures were encountered.²⁴ In addition, both an MVD and rhizotomy were performed for 56 patients in which vascular compression did not cause nerve indentation or distortion. Overall, about 75% of patients had an excellent result and 8% had good results. They reported increased sensory loss and dysesthesias in the rhizotomy group, particularly those with a history of prior lesioning or lysis procedures. At 5-year follow-up, they found a nonsignificant trend toward a better outcome in patients who underwent both a MVD and rhizotomy and a worse outcome for rhizotomy alone.

In Klun's series, immediate pain relief was achieved in 86% of the PSR

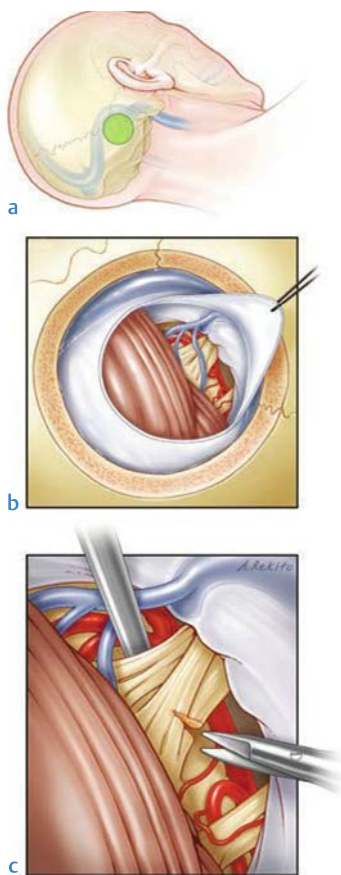


Fig 12.1 (a) Keyhole microvascular decompression craniotomy at the junction of the transverse and sigmoid sinuses. (b) Dural retraction showing the cerebellopontine angle and cranial nerve 5. (c) Inferocaudal partial sensory rhizotomy with microscissors.

patients; however, this rate dropped to 84% during the follow-up period.²⁷ The recurrence rate difference between the MVD group and rhizotomy group was dramatic because only 6% of the MVD group recurred versus 49% in the rhizotomy group. Complications were uncommon and consisted of hearing loss, corneal reflex impairment, and lesioning of the portio minor. The degree of parasthesias among those in the rhizotomy group varied widely; however, painful dysesthesias or anesthesia dolorosa was not encountered. One interesting point from his series was that labial herpes occurred in about half of the MVD patients and in virtually all of the PSR patients.

Lui and Apfelbaum described their experience with partial sensory rhizotomy of 40 patients³ and reported complete pain relief in 32 of the patients (80%). In the 8 patients who did not have complete resolution of their pain, either medical treatment or additional percutaneous lesioning procedures helped control all but 1 patient's pain. Delitala et al reported immediate complete pain relief in 88% of their 42 patients.²⁵ During a 2-year follow-up this rate dropped to 72%; however, no patient had significant parasthesias. The authors recommended that venous compression and arterial compression that did not distort the nerve be considered a negative exploration and these patients should undergo partial sensory rhizotomy. In the small series of eight patients, five had complete pain relief without recurrence.²⁶ Two patients recurred at 2 and 3 years, respectively, and the pain was controlled medically. One patient had pain greater than was experienced preoperatively, and one patient developed anesthesia dolorosa. No hearing loss, corneal reflex deficits, or facial weakness was noted. In the "nerve combing" series of 10 patients, the overall pain-free outcome at 3 years was 70%, lower than the expected MVD pain-free outcome rate.³³

■ Indications and Technique

Since 2011 the senior author has modified the standard microscopic MVD and has performed a fully endoscopic MVD.^{40–42} We believe the use of the neuroendoscope for posterior fossa pathology may reduce complications from traditional microscopic surgery, leading to improved outcomes. With the endoscope, minimal retraction on the cerebellum is needed for visualization, and the neuroendoscope allows superior panoramic visualization compared with a traditional microscope. In addition, the endoscope provides an unobstructed view of the Obersteiner–Redlich zone at the brainstem as well as at the entry into Meckel cave, and may provide additional detail of vascular compression (**Figs. 12.2, 12.3, 12.4**). This panoramic view may also be useful for evaluating which trigeminal nerve fascicles to incise during a rhizotomy.

In a quick review of patients of the senior author undergoing microvascular decompression within a 1-year span (May 2012–May 2013), 41 patients underwent posterior fossa exploration. Of these, 6 patients underwent microscopic surgery because of lack of endoscope sterilization at time of procedure. The vast majority, 85.3% of patients (35 of 41), underwent purely endoscopic surgery. Eleven of the 41 patients (27%) underwent a neurolysis with a round knife along the fascicles of the nerve similar to the "nerve combing" technique described above. Neurolysis (a variant of partial sensory rhizotomy) was performed when no compressive vessel was identified or when the senior author felt that the degree of compression by ar-

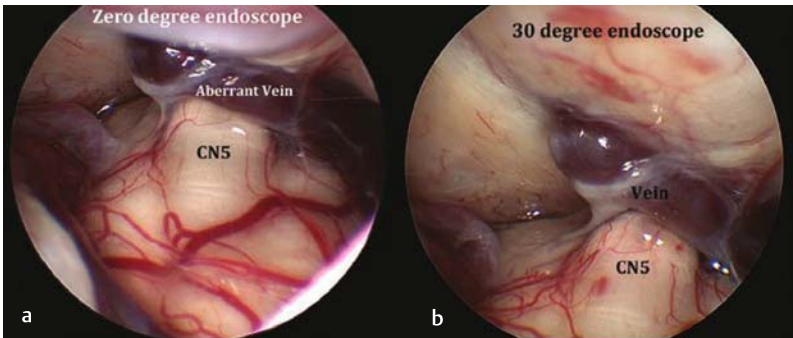


Fig. 12.2 Venous compression. This is a right-sided microvascular decompression for trigeminal neuralgia. One of the benefits of the angled endoscope is that it can help the surgeon appreciate anatomy that is not visible with a microscope. **(a)** In this case, the 0-degree endoscope (standard microscopic view) provides only a head-on view of the trigeminal nerve at the root entry zone. **(b)** A 30-degree up-angled endoscope can be used to carefully delineate the petrosal vein crossing the distal aspect of the trigeminal nerve. CN, cranial nerve.

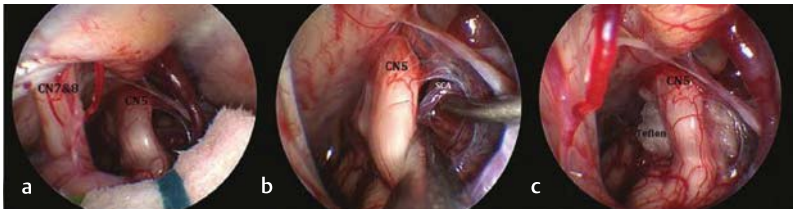


Fig. 12.3 SCA (superior cerebellar artery) compression. This is a left-sided microvascular decompression of trigeminal neuralgia. **(a, b)** This is an example of an endoscopic view of the classic vascular compression by the SCA, resulting in lateral and inferior deformation of the trigeminal nerve (CN 5). The Dandy petrosal vein is not sacrificed in the great majority of fully endoscopic procedures because the panoramic view of the endoscope allows the vein to be visualized throughout the procedure. The degree of stretch on the vein can be monitored at all times. **(c)** Notice the improvement in the coloration of the nerve after the Teflon is placed in between the trigeminal nerve and the superior cerebellar artery. CN, cranial nerve.

tery or vein was not great enough to be the sole cause of pain. Short-term pain-free outcomes are encouraging. The similar rates of neurolysis in our fully endoscopic series and in the PSR series in the literature review may suggest that even with angled endoscopes, there is no increase in the ability to identify additional vascular compression. However, longer term studies are needed.

For other facial neuralgias that require a neurectomy, such as geniculate neuralgia, our experience is that the endoscope allows a much greater appreciation of the anatomy and identification of the nerve. For geniculate neuralgia, the classic micro-

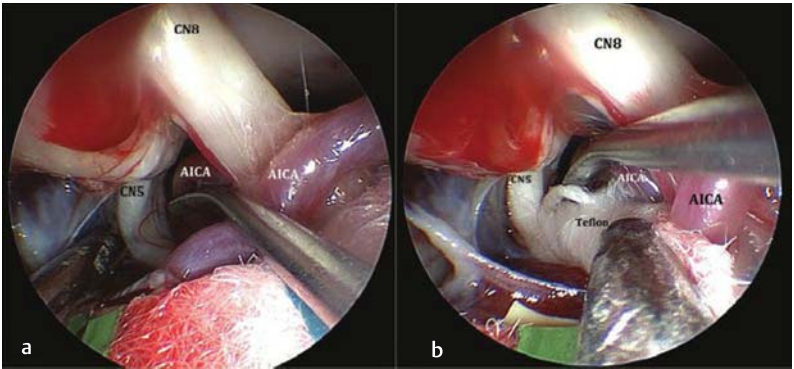


Fig. 12.4 AICA (anterior inferior cerebellar artery) compression. This is a right-sided microvascular decompression for trigeminal neuralgia. In this case, the vascular compression is easily identified. **(a)** The fifth nerve is deformed from the caudal side by a branch of the AICA. **(b)** A single piece of Teflon is placed between the trigeminal nerve and AICA. CN, cranial nerve.

scopic maneuver is to retract at the flocculus below the eighth nerve near the ninth nerve and to search for a distinct geniculate nerve. However, using a 30-degree endoscope facing medially eliminates the need for a retractor. **Fig. 12.5a** shows that using a 0-degree endoscope allows visualization of the seventh nerve but not the nervus intermedius. The 30-degree angled-down endoscope (**Fig. 12.5b, c**) allows much better appreciation of the anatomy and identification of the nervus intermedius. In the senior author's experience, occasionally the endoscope has illuminated vascular compression that could not be visualized using a traditional microscope. In these cases, the endoscope was essential in avoiding a sensory rhizotomy.

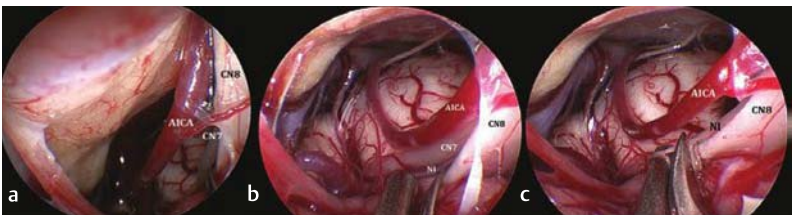


Fig. 12.5 Geniculate neuralgia. The geniculate nerve can be very difficult to find. **(a)** The 0-degree endoscope is the standard microscopic view, which allows visualization of the seventh nerve, but not of the nervus intermedius (NI). **(b)** However, a 30-degree endoscope eliminates the need for a retractor and allows much better appreciation of the anatomy and identification of the nervus intermedius. **(c)** The sectioning of the nervus intermedius. AICA, anterior inferior cerebellar artery; CN, cranial nerve.

■ Conclusion

In the advancement of the surgical treatment of trigeminal neuralgia there have been a number of improvements of surgical technique. The history of trigeminal rhizotomy ranges from the complete removal of the gasserian ganglion, to complete sectioning of the dorsal root, to preserving the motor and ophthalmic fibers as well as differentiating between the mandibular and maxillary branching fibers. The current practice involves posterior fossa exploration with performance of a standard Jannetta MVD procedure if the vascular contact appears to be significant. The partial sensory rhizotomy is performed at a rate of approximately 28%. The rhizotomy is generally performed on the infero-caudal-lateral portion of the nerve using a range of techniques. From the literature reports, a large proportion of patients who undergo this procedure have good pain relief with only partial sensory loss. The lack of “satisfaction” of patients undergoing partial sensory rhizotomy may be a reflection of sensory complications or a reflection of underlying difference in pain etiology. Fully endoscopic techniques may identify additional points of vascular contact, but outcomes remain preliminary. Partial sensory rhizotomy of the trigeminal nerve continues to be a tool in the armamentarium of surgeons treating patients with trigeminal neuralgia. Although the level of evidence is Class III, PSR procedures should be used in a judicious manner.

Editor's Comments

Partial sensory rhizotomy (PSR) is an important aspect of the surgical treatment of trigeminal neuralgia (TN). It has not gotten nearly as much attention as microvascular decompression (MVD), but if 28% of patients who undergo surgery for planned MVD have PSR due to the absence of convincing vascular compression, then it should have. Parenthetically, we have found almost *exactly* the same rate of no vascular compression in our series of posterior fossa surgery for TN.

Doctors Lang and Lee have outlined the history of PSR and the current state of the outcome data. Loyalty to their university tradition, and to the contributions of Spiller and Frazier, may have prevented them from acknowledging the seminal contribution of Harvey Cushing to the development of trigeminal rhizotomy.⁴³ They have summarized the literature on rhizotomy, although, as they point out, the evidence derives only from case series (Class III).

Whether a PSR is performed by microscopic or endoscopic techniques is a matter that could easily be subject to a randomized trial, at centers adept at both techniques. In my view, our principal goal should be a randomized prospective trial of MVD versus PSR for trigeminal neuralgia.

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13

Trigeminal Neurectomy

Richard K. Osenbach

Painful conditions involving structures of the face and teeth have been recognized for more than 10 centuries. Indeed, early in the previous millennium, the Persian physician and philosopher Avicenna wrote about painful disorders involving the face. Facial pain is, in fact, a common affliction that affects many people. In most cases, the symptoms are acute and transient and resolve with minimal medical intervention. In some patients, however, facial pain may persist and evolve into a disabling chronic pain syndrome that can be quite refractory to conventional pain therapies.

The best-known chronic facial pain syndrome is classic trigeminal neuralgia. Although John Locke has been credited with the description of this malady in 1677, the first clear description actually was given in 1671 by Drs. Johannes Michael Fehr and Elias Schmidt, who elucidated the details of the ailment as it affected Johannes Laurentius Bausch, a physician, philosopher, and municipal counselor of Schweinfurth, Franconia.¹

Although trigeminal neuralgia is the most-recognized and best-studied of the facial neuralgias, it is by no means the only cause of facial pain. Indeed, many conditions can result in chronic facial pain of such severity as to warrant medical intervention. Some of these conditions may cause damage or injury to one or more peripheral branches of the trigeminal nerve and lead to a condition known as *trigeminal neuropathic pain*.² For example, surgery on the maxillary sinus can be complicated by damage to the infraorbital nerve, which in turn can lead to neuropathic pain in the corresponding cutaneous distribution of the nerve. Facial fractures involving the supraorbital ridge can lead to damage to the supraorbital and/or supratrochlear nerves. Also, dental procedures such as root canal and tooth extractions can damage the peripheral nerve endings and lead to chronic intractable pain in the jaw and mouth.

The treatment of facial pain in general and trigeminal neuralgia in particular has undergone considerable evolution in terms of both medical and surgical therapy. Currently, an array of procedures can be performed for medically refractory trigeminal neuralgia as well as for other selected facial neuralgias. This chapter is devoted to examining the indications for, techniques of, and results from peripheral trigeminal neurectomy, the oldest recorded treatment for trigeminal neuralgia. These particular techniques are currently more of historical than practical interest, given the alternatives such as the minimally invasive percutaneous needle techniques (radiofrequency gangliolysis, glycerol rhizolysis, and balloon microcompression) and stereotactic radiosurgery, especially for the treatment of trigeminal neuralgia. Moreover, the surgical treatment of trigeminal neuropathic pain has evolved with the increasing application of nonablative techniques such as peripheral trigeminal branch stimulation and epidural motor cortex stimulation. Regardless, even

though these techniques are rarely if ever utilized in contemporary neurosurgical practice, they still have an important place in the history of neurosurgical treatment of trigeminal neuralgia and other facial pain disorders.

■ History of Peripheral Trigeminal Branch Ablation

The destruction of peripheral branches of the trigeminal nerve as a treatment for trigeminal neuralgia goes back more than two centuries. Schlichting was originally credited with performing the first peripheral nerve operation for trigeminal neuralgia in 1748. As it turns out, the first peripheral destructive operations for *tic douloureux* were carried out, albeit unsuccessfully, by Maréchal, surgeon to King Louis XIV, in 1730 and 1732.³ In the second patient, pain relief was achieved temporarily, but it lasted only 2 months. Temporary pain relief from peripheral destructive procedures would prove to be a common, recurring theme. André reoperated on Maréchal's second patient by exposing the mandible and then applying a hot iron to exfoliate the bone. He then enlarged the mandibular foramen with a trephine, presumably to expose the inferior alveolar nerve, which he destroyed by applying a liquid caustic.

Following Maréchal and André, others performed a variety of procedures on peripheral branches of the trigeminal nerve for the treatment of trigeminal neuralgia. Lizars and Warren cut the inferior alveolar nerve within its bony canal of the mandible. Malgaige, in 1849, and Langenbeck, some two decades later, devised a method of sectioning the infraorbital nerve on the floor of the orbit. Presumably because of the transient pain relief with the more distal procedures, attempts were made to divide the nerve(s) as close to the trigeminal ganglion as possible. Indeed, Kronlein devised a series of procedures for exposing the maxillary and mandibular divisions at the foramen rotundum and foramen ovale, respectively. Kronlein's technique involved making a U-shaped incision extending from the ear to the molar prominence and then resecting the zygoma and coronoid process of the mandible. He then reflected the temporalis muscle superiorly to expose the second and third divisions exiting the cranial base.⁴

During the same period, it was discovered that injection of a destructive liquid into branches of the trigeminal nerve was not only effective but also simpler than open ablative procedures. Numerous substances were tried, including chloroform, osmic acid, 2% cocaine followed by 60% ethanol, chromates, formalized glycerine, carbolyzed glycerine, alcoholized metholated glycerine, ether, antipyrine, salicylate of soda, and quinine salts in various proportions and doses.¹ Sicard, in 1918, having tried many of these agents, concluded that alcohol was the best compound. In addition to chemical neurolysis, several surgeons attempted peripheral trigeminal ablation using radiofrequency electrocoagulation.

Ultimately, as experience accumulated, it became clear that the relief provided by peripheral branch ablation was only temporary and that developing procedures that could produce more successful long-term relief was desirable. Although more sophisticated procedures currently exist (as discussed in other chapters), peripheral branch destruction remains a useful procedure in the appropriate clinical setting (see subsequent discussion).

■ Clinically Relevant Anatomy of the Peripheral Trigeminal Nerve

A thorough understanding of the anatomy and sensory distribution of the peripheral trigeminal nerve is essential when considering any type of ablative procedure on one or more of these nerves. Indeed, failure to recognize correctly the neural distribution of the patient's nerve consists of three major divisions: the ophthalmic (V-1), maxillary (V-2), and mandibular (V-3) nerves. Phylogenetically, the trigeminal nerve is the main cutaneous sensory nerve of the head and face. As the phylogenetic ladder is ascended, the sensory distribution of the trigeminal nerve is expanded, whereas those of the facial, glossopharyngeal, and vagal nerves (of which the afferent inputs enter the spinal trigeminal tract and nucleus) are reduced. In addition to providing most of the somatic sensory input from cutaneous structures, the trigeminal nerve supplies sensation to the cornea, most of the mucous membrane surfaces of the oral and nasal cavities, the mucosa of the paranasal sinuses, the intracranial dura, periodontal structures, and portions of the large intracranial arteries.⁵ The pertinent anatomy of each division of the trigeminal nerve is described later in this chapter. The cutaneous innervation of the head is illustrated in **Fig. 13.1**.

Ophthalmic Nerve

The ophthalmic nerve, or first division, is a flat band, approximately 2.5 cm long, that exits the anterior superior portion of the gasserian ganglion and enters the

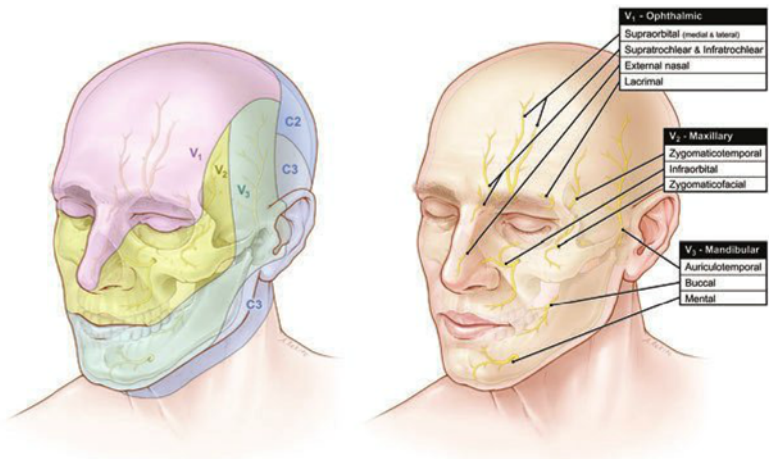


Fig. 13.1 The cutaneous innervation of the head and neck. The cutaneous branches of the major divisions of the trigeminal nerve are indicated. Note that the supraorbital, infraorbital, and mental nerves all lie in the vertical plane of the pupil.

orbit through the superior orbital fissure. The ophthalmic nerve is purely sensory, providing sensation to the globe, conjunctiva, lacrimal gland, mucous membranes of the nose and paranasal sinuses, and the skin of the forehead, scalp, eyelids, and nose. The ophthalmic nerve sends a small recurrent filament to supply the tentorium and dura of the anterior fossa and then, before passing through the superior orbital fissure, divides into three branches: frontal, lacrimal, and nasociliary.

Frontal Nerve

For all practical purposes, the frontal nerve, which is the largest of the three branches, can be considered a continuation of the ophthalmic nerve. The nerve passes forward and, at approximately the midpoint from its origin to the superior orbital rim, divides into the larger *supraorbital* and smaller *supratrochlear* nerves. The supraorbital nerve leaves the orbit through the supraorbital foramen, providing filaments to the upper eyelid in the process. It then divides into medial and lateral branches beneath the frontalis muscle. The smaller, medial branch pierces the frontalis muscle and supplies cutaneous innervation to the scalp posterior to the parietal bone. The larger, lateral branch penetrates the galea and supplies the scalp all the way back to the lambdoid suture. Occasionally, the supraorbital nerve branches proximal to its foramen, in which case the lateral branch generally occupies the foramen and the medial branch exits through a separate foramen.

The supratrochlear nerve turns medially within the orbit above the pulley of the superior oblique muscle, where it produces a filament that communicates with the infratrochlear branch of the nasociliary nerve. After piercing the orbital fascia, branches are given off to supply the skin and conjunctiva of the medial upper lid. After exiting its foramen, it divides into branches that supply the lower and medial portions of the forehead.

Lacrimal Nerve

The *lacrimal nerve* is the smallest of the three branches arising from the ophthalmic nerve. It passes anteriorly, entering the orbit through the narrowest portion of the superior orbital fissure. Within the orbit, it runs along the superior border of the lateral rectus muscle and enters the lacrimal gland to supply the gland and the adjacent conjunctiva. The nerve then pierces the orbital septum and terminates in the skin of the upper eyelid.

Nasociliary Nerve

Intermediate in size between the frontal and lacrimal nerves, the *nasociliary nerve* occupies the deepest position in the orbit of the three branches. The nerve enters the orbit between the heads of the lateral rectus muscle and between the superior and inferior divisions of the oculomotor nerve. The nasociliary nerve runs obliquely through the orbit, crossing above the optic nerve and below the superior rectus and oblique muscles approaching the medial wall of the orbit. At this point, it exits the orbit through the anterior ethmoidal foramen as the anterior ethmoidal nerve and enters the intracranial cavity just above the cribriform plate, following which it penetrates the bone at the side of the crista galli to enter the nasal cavity, where it supplies branches to the mucous membranes of the nose.

The nasociliary nerve gives rise to several branches. The *long ciliary nerves* emerge as the nasociliary nerve crosses the optic nerve. The long ciliary branches pass through the ciliary ganglion and accompany the short ciliary nerves, pierce the posterior sclera, and, running between it and the choroid, supply sensation to the iris and cornea. The *ethmoidal branches* supply the mucosa of the sinuses. These consist of a *posterior ethmoidal branch*, which supplies the posterior ethmoid and sphenoid sinuses, and *anterior ethmoidal branches*, which arise as the nerve passes through the anterior ethmoidal foramen; the latter supply the frontal and anterior ethmoid sinuses.

The infratrochlear nerve arises from the nasociliary nerve just proximal to the anterior ethmoidal foramen. After running anteriorly along the upper border of the superior rectus muscle, it is joined by a branch of the supratrochlear nerve and then passes to the medial angle of the eye to supply the skin of the eyelids and side of the nose, the conjunctiva, lacrimal sac, and caruncula lacrimalis. Finally, internal and external nasal branches are given off: The *internal nasal branches* supply the mucosa of the anterior part of the septum and lateral wall of the nasal cavity; the *external nasal branches* supply the skin of the ala and apex of the nose.

Maxillary Nerve

The *maxillary nerve*, which is entirely sensory, arises from the midportion of the trigeminal ganglion. It initially passes rostrally in the inferior portion of the lateral wall of the cavernous sinus, continues beneath the dura, and exits the skull through the foramen rotundum. Henceforth, it courses through the pterygopalatine fossa, enters the orbit through the inferior orbital fissure, and then runs along the floor of the orbit (roof of the maxillary sinus). In the posterior portion of the orbit, it becomes the infraorbital nerve, which then continues in the infraorbital groove and ultimately emerges onto the face through the infraorbital foramen. The maxillary nerve provides cutaneous innervation to the middle of the face, lower eyelid, side of the nose, and the upper lip. It also supplies sensation to the mucous membranes of the nasopharynx, maxillary sinus, soft palate, tonsil, and roof of the mouth as well as the upper gums and teeth. There are five sets of branches of the maxillary nerve: intracranial, pterygopalatine, posterior superior alveolar, infraorbital, and facial.⁵

Intracranial and Pterygopalatine Branches

The *middle meningeal nerve* is an intracranial branch of the maxillary nerve, which arises just distal to the gasserian ganglion. The nerve accompanies the middle meningeal artery and supplies the dura. There are multiple branches within the pterygopalatine fossa, including the zygomatic nerve, pterygopalatine nerves, and posterior superior alveolar branches. The *zygomatic nerve* originates in the pterygopalatine fossa, enters the orbit through the inferior orbital fissure, and divides into temporal and facial (malar) branches. The *temporal branch*, after running in the groove within the zygoma along the lateral orbital wall, pierces the zygoma and enters the temporal fossa, where it penetrates the temporalis muscle and is distributed to the skin of the side of the forehead. The *facial* or *malar branch* emerges from the inferolateral angle of the orbit, pierces the orbicularis oculi muscle, and supplies the skin of the malar eminence.

The *pterygopalatine nerves* are divisible into four groups: orbital, palatine, posterior superior nasal, and pharyngeal. The *orbital (ascending) branches* enter the orbit through the inferior orbital fissure to supply the periosteum, along with the posterior ethmoid and sphenoid sinuses, through filaments that pass through the frontoethmoidal suture. The *greater (anterior) palatine nerve* exits the fossa through the pterygopalatine canal and enters the hard palate to supply the gums and mucosa of the hard palate nearly as far as the incisors. The greater palatine nerve has two sets of branches: the *lesser palatine nerves*, which emerge from their corresponding foramen and supply the soft palate, uvula, and tonsil; and the *posterior inferior nasal branches*, which are distributed to the inferior nasal turbinate. The *posterior superior nasal branches* enter the posterior nasal cavity through the sphenopalatine foramen and supply the superior and middle turbinates, the mucosa of the posterior ethmoid sinuses, and the posterior nasal septum. The *pharyngeal branch (pterygopalatine nerve)* passes through the pharyngeal canal to supply the mucosa of the nasopharynx posterior to the meatus of the eustachian tube.

Posterior Superior Alveolar Branches

The *posterior superior alveolar branches* arise from the trunk of the maxillary nerve just before it enters the infraorbital groove. These branches radiate several twigs, which supply the gums and adjacent mucosa before entering the posterior alveolar canals. They then traverse the bony maxilla and provide innervation to the molar teeth along with branches to the maxillary sinus. The posterior superior alveolar branches communicate with the middle superior alveolar nerve (discussed later).

Branches in the Infraorbital Canal

The maxillary nerve has two main branches within the infraorbital canal: anterior and middle superior alveolar branches. The *middle superior alveolar branch* supplies the two premolar teeth; the *anterior superior alveolar branch* supplies the incisor and canine teeth. After giving rise to these branches, the infraorbital nerve emerges into the face through the infraorbital foramen.

Facial Branches

The *inferior palpebral branches* pass superiorly and supply the skin and conjunctiva of the lower eyelid, anastomosing with the zygomaticofacial nerves at the lateral angle of the orbit. The *external nasal branches*, which communicate with the terminal twigs of the nasociliary nerve, innervate the skin of the nose and the cartilaginous septum. The largest facial branches in both size and number are the *superior labial branches*, which pass deep to the levator labii superioris muscle and supply the skin of the upper lip, the mucous membranes in the mouth, and the labial glands.

Mandibular Nerve

The *mandibular or third division* is the largest of the peripheral trigeminal branches. In addition to the large sensory root, the mandibular nerve contains a small motor root. The afferent portion of the nerve supplies the skin of the temporal region,

auricula, external meatus, cheek, lower lip, and lower part of the face. The mucous membranes of the cheek, tongue, and mastoid air cells along with the lower teeth and gums are also supplied by the third division, along with the mandible and temporomandibular joint. Portions of the dura and skull also receive sensory supply from this division. The sensory and motor roots emerge separately from the skull base through the foramen ovale, but they unite just outside the skull, forming a common trunk for a distance of 2 or 3 mm. The main trunk gives off a meningeal branch and the medial pterygoid nerve before bifurcating into a smaller anterior and larger posterior division.

Anterior Division

The *anterior division* of the mandibular nerve contains a small number of sensory fibers along with all the motor fibers except for those carried in the medial pterygoid and mylohyoid nerves. The *masseteric nerve* passes laterally above the lateral pterygoid to enter the masseter muscle and then provides a twig to the temporomandibular joint. The *buccal nerve* passes between the heads of the lateral pterygoid muscle and eventually emerges from beneath the inferior border of the masseter. It supplies the skin of the cheek over this muscle along with penetrating branches, which supply the mucosa of the mouth and gums in this area.

Posterior Division

The *posterior division* of the mandibular nerve has three major branches: the auriculotemporal, lingual, and inferior alveolar nerves. The posterior division is chiefly sensory, although a small number of motor fibers also travel in the branches.

The *auriculotemporal nerve* typically originates from two roots, which encircle the middle meningeal artery in the vicinity of the foramen spinosum. It passes posteriorly, deep to the lateral pterygoid muscle along the medial aspect of the mandible, and then it turns superiorly and runs with the superficial temporal artery between the auricula and mandibular condyle. After it emerges from beneath the parotid gland, the nerve passes over the root of the zygoma and divides into superficial branches. The small branches of the auriculotemporal nerve provide afferent innervation to the skin of the temporal region (superficial temporal branch), the skin of the anterior auricula (primarily the helix and tragus), the external auditory meatus, and the temporomandibular joint.

The *lingual nerve* initially lies deep to the lateral pterygoid muscle and runs parallel to the inferior alveolar nerve, with which it has an anteromedial relationship. It then runs between the mandible and medial pterygoid muscle and crosses obliquely above the superior pharyngeal constrictor and styloglossus to reach the lateral aspect of the tongue. After passing between the hyoglossus and the submandibular gland, the lingual nerve runs along the undersurface of the tongue, providing somatic sensation to the anterior two thirds of the tongue and the adjacent mucous membranes of the mouth and gums.

After arising from the posterior division of the mandibular nerve, the *inferior alveolar (inferior dental) nerve* accompanies its corresponding artery, running deep to the lateral pterygoid and then passing between the sphenomandibular ligament and ramus of the mandible to enter the mandibular foramen. It travels anterior within the mandible in the mandibular canal to the mental foramen

and divides into two terminal branches: the *mental* and *incisive nerves*. The dental branches arise from the main trunk of the inferior alveolar nerve within the bone and supply the lower molar and premolar teeth. The incisive branches form a plexus, which supplies the lower canine and incisor teeth. After exiting through the mental foramen, the mental nerve divides into three branches, which supply the skin on the chin and the skin and mucous membrane of the lower lip.

■ Indications for Peripheral Trigeminal Neurectomy

It is a well-known fact that destructive procedures on the peripheral branches of the trigeminal nerve—in particular, peripheral branch neurectomies—are effective in producing pain relief from trigeminal neuralgia. Once common, these procedures currently are rarely if ever performed for trigeminal neuralgia because of the popularity of microvascular decompression and percutaneous retrogasserian techniques, such as radiofrequency thermocoagulation, glycerol injection, and balloon microcompression. Additionally, it seems as though stereotactic radiosurgery continues to play an ever-increasing role in the treatment of trigeminal neuralgia. Indeed, some surgeons now advocate stereotactic radiosurgery as primary treatment for trigeminal neuralgia. Nevertheless, the reason that peripheral neurectomy is effective is it eliminates the nociceptive afferent input to the spinal trigeminal nucleus and tract. In addition, there is evidence that trauma to peripheral branches of the trigeminal nerve produces temporary degenerative changes in trigeminal ganglion cells, which may contribute to the pain relief that occurs following neurectomy. The principal reason that peripheral neurectomy is not as popular as it once was is that it is only a temporary solution to the problem.

Although purely palliative, peripheral neurectomy has a number of advantages. The fact that peripheral neurectomy is capable of producing pain relief is unquestionable.⁶⁻¹¹ Peripheral neurectomies are generally technically simple as long as one has an understanding of the pertinent anatomy. These procedures can be performed under general or local anesthesia. One of the most important advantages is that peripheral neurectomy is a low-morbidity procedure that can be especially beneficial in an older and infirm patient who might not tolerate a more involved procedure. It is particularly useful in patients who suffer from trigeminal neuralgia involving the first division because there is no risk of producing corneal anesthesia, as there is with retrogasserian procedures, especially radiofrequency lesions. Peripheral neurectomy also may prove beneficial in the treatment of selected patients with trigeminal neuropathic pain syndromes caused by dental surgery, surgery on the paranasal sinuses, or injury to peripheral branches from facial trauma.

In patients with trigeminal neuropathic pain, several factors may have prognostic value in selecting patients who might benefit from a neurectomy. These factors are similar to those used to select appropriate candidates with peripheral nerve neuropathic pain for neurectomy or neuroma resection. These factors include pain related to trauma, pain within a single nerve distribution, the presence of Tinel sign, and complete pain relief with local anesthetic nerve blockade.^{12,13} If all these conditions are satisfied, neurectomy is 50 to 60% effective in relieving pain. One final advantage is the lack of need for any special intraoperative imaging

such as biplane fluoroscopy, which is essential in performing retrogasserian procedures. As with any surgical procedure being considered for intractable pain, all patients who are candidates for peripheral neurectomy should have completed an exhaustive course of pharmacological therapy that failed to bring relief.

■ Peripheral Neurolysis Using Injection Techniques

Injection techniques play an important role in the management of patients with refractory facial pain syndromes, especially when a peripheral ablative procedure is contemplated. Indeed, local anesthetic nerve blocks can have excellent predictive value in determining whether a neurectomy will be beneficial. An alternative to surgical neurectomy is the injection of neurolytic substances into the nerve. The most commonly used agent has traditionally been alcohol, although within the last decade, there have been reports regarding the use of other agents such as streptomycin.^{14,15}

Peripheral trigeminal branch blocks also can assist in illuminating the exact neural distribution of pain. In the performance of diagnostic blocks, several practical points should be kept in mind. The first is the choice of local anesthetic. Generally, an agent such as 0.5% lidocaine or 0.25% bupivacaine is satisfactory. My preference is to use a 50:50 mixture of 1% lidocaine and 0.5% bupivacaine *without* epinephrine. Under normal circumstances, primary afferent neurons do not exhibit catecholamine sensitivity, and their activity is unaffected by catecholamines or sympathetic outflow.¹⁶ Afferent neurons that have been injured, however, exhibit upregulation of catecholamine receptors and develop hypersensitivity to catecholamines. Under these circumstances, injection of a local anesthetic containing epinephrine around an injured nerve actually may potentiate pain and conceivably produce a false-negative (no pain relief when pain relief may have occurred) result from the block. Another important point is that a diagnostic nerve block should be as precise and specific as possible such that accurate information is gained. This is accomplished by having a thorough understanding of the anatomy and by using as little volume of anesthetic as possible. Injection of large amounts of anesthetic may result in anesthesia in the desired nerve distribution but may also diffuse and result in blockade of other nerves and thereby skew the information. The following section outlines the techniques for somatic blockade of the peripheral trigeminal branches of the head.

Ophthalmic Nerve Branches

Supraorbital and Supratrochlear Nerves

Local anesthetic blockade of the supraorbital and supratrochlear nerves is a simple procedure. The supraorbital nerve and foramen lie in the vertical place occupied by the pupil with the patient looking straight ahead.¹⁷ The block is achieved most easily above the eyebrow after the nerve has exited its foramen. The supratrochlear nerve runs parallel and approximately one fingerbreadth medial to the supraorbital nerve. The nerve can be blocked as it emerges from the eyebrow or by medial extension of an anesthetic wheal used to block the supraorbital nerve (**Fig. 13.2**).

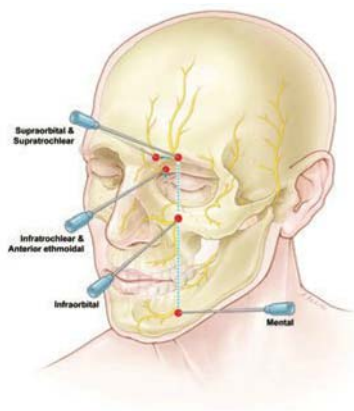


Fig. 13.2 The entry sites and needle trajectories for blockade of the superficial branches of the trigeminal nerve.

Infratrochlear and Anterior Ethmoidal Nerves

The terminal branches of the nasociliary nerve can be blocked by inserting a 27-gauge needle 1 cm above the inner canthus and directing it backward and slightly medially to a depth of approximately 1 inch (**Fig. 13.2**). This trajectory allows the needle to pass just lateral to the medial wall of the orbit but medial to the globe and medial rectus muscle. Once positioned, 1 mL of local anesthetic is slowly injected as the needle is withdrawn. The most significant complication is related to intraorbital hemorrhage from damage to the orbital veins, which can result in proptosis. Therefore, a small-gauge needle should be used, and repeated insertion should be avoided.

Maxillary Nerve and Branches

Main Trunk

The main trunk of the maxillary nerve can be blocked within the pterygopalatine fossa using a lateral approach (**Fig. 13.3**). Blockade of the nerve at this point will produce profound analgesia of the ipsilateral upper jaw, teeth, and overlying skin and soft tissues. The point of needle insertion is at the midpoint of the zygoma overlying the coronoid notch of the mandible. The needle is introduced and directed medially until contact is made

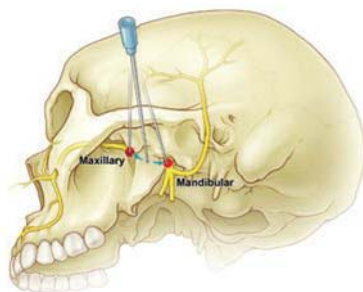


Fig. 13.3 The technique for blockade of the maxillary and mandibular nerves from a lateral approach.

with the lateral pterygoid plate along the medial wall of the infratemporal fossa, usually at a depth of about 5 cm. The needle then is “walked” along the lateral pterygoid plate anteriorly until the pterygopalatine fossa is encountered, and then it is advanced a centimeter deeper.¹⁷ A few milliliters of local anesthetic are instilled to produce the desired effect. Incidentally, this technique also can be used for blockade of the sphenopalatine ganglion, although selective blockade of the ganglion without involving the maxillary nerve is difficult if not impossible. The major morbidity of maxillary nerve block is a

hematoma in the infratemporal or pterygopalatine fossa, which can spread into the orbit and produce a black eye. Treatment is according to symptoms. The other possible complication is related to temporary blindness due to diffusion of local anesthetic. Because of the proximity of this region to the orbit, this procedure probably should be avoided for permanent neurolytic blockade.

Infraorbital Nerve

Blockade of the infraorbital nerve can be performed at the junction of the medial and middle thirds of the inferior orbital rim. The landmark for the infraorbital nerve is again the pupillary line. The infraorbital foramen is configured such that its long axis is directed medially and caudally. Therefore, cannulation of the foramen requires that the needle be directed laterally and cephalad (**Fig. 13.2**). The block can be accomplished by injecting 1 to 2 mL of anesthetic using a small-gauge needle at the point where the nerve exits the foramen.

Mandibular Nerve and Branches

Main Trunk

The main trunk of the mandibular nerve can be accessed using the same approach as that described for the maxillary nerve in the pterygopalatine fossa. The technique differs once the medial wall of the infratemporal fossa is encountered. The needle is “walked” *posteriorly* along the lateral pterygoid plate until paresthesias in the third division are elicited (**Fig. 13.3**).¹⁷ Once paresthesias are obtained, several milliliters of local anesthetic are instilled. If a high enough concentration of local anesthetic is used (1% lidocaine or its equivalent), motor blockade will likely occur. Although this is not particularly problematic with temporary blockade, permanent neurolysis can result in a lack of coordination of jaw movements, which can be extremely distressing to the patient. Another consideration is the proximity of the otic ganglion, which supplies secretomotor fibers to the parotid gland to the mandibular nerve. Mandibular blockade cannot be accomplished without anesthetizing the otic ganglion. Thus, permanent mandibular neurolysis would result in permanent impairment of parotid secretion. One final word of caution regarding third-division blockade: Once the needle has been “walked” posteriorly off the lateral pterygoid plate, it should never be inserted deeper because it can penetrate the superior constrictor muscle and enter the pharynx.

Mental and Auriculotemporal Nerves

The mental foramen lies in the same vertical plane as do the pupil and the supraorbital and infraorbital foramina. The position varies depending on age and dentition, lying more caudal on the mandible in younger people and nearer the margin of the mandible in older and edentulous patients. To perform an extraoral block of the mental nerve, the needle is directed anteriorly and caudally.¹⁷ The auriculotemporal nerve can be blocked as it ascends over the posterior root of the zygoma accompanied by the superficial temporal artery, which lies anteriorly.

■ Nerve Avulsion (Neurectomy) Procedures

Supraorbital Neurectomy

Supraorbital neurectomy is indicated for patients with trigeminal neuralgia limited to the first division or for patients with posttraumatic neuropathic pain within the distribution of an accessible nerve. The technique of supraorbital neurectomy is illustrated in **Fig. 13.4**. My preference is to perform the procedure under local anesthesia, although if it is tolerated, general anesthesia also can be used. The patient is positioned supine with the head supported on a padded headrest and the back of the table is raised to a position of comfort. The eyebrow should

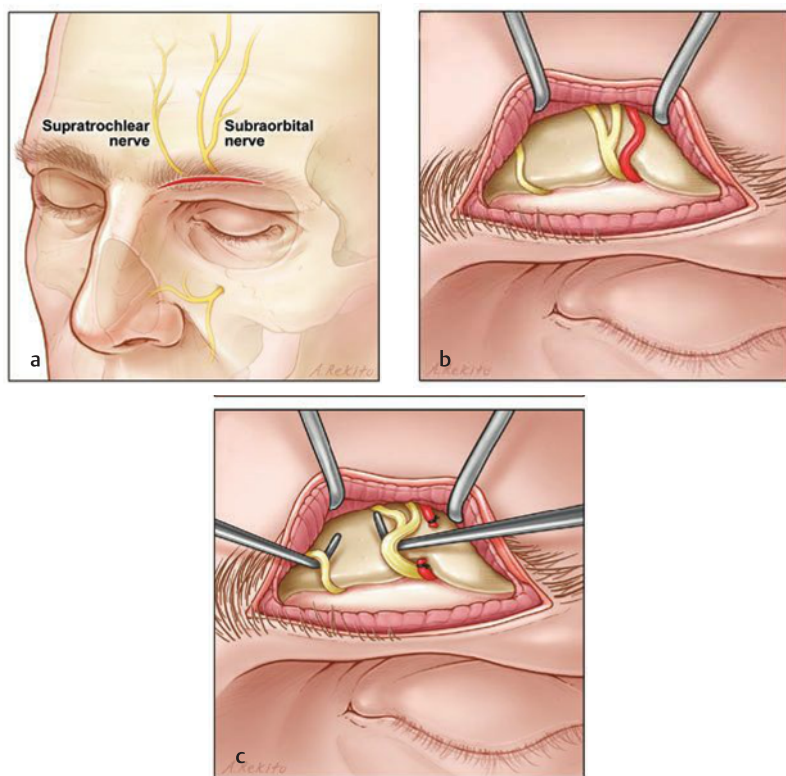


Fig. 13.4 Exposure of the supraorbital and supratrochlear nerves for the purpose of neurectomy via avulsion or ligation/division. **(a)** Skin incision through the eyebrow relative to surface anatomy and underlying nerves. **(b)** Dissection through orbicularis oculi muscle to expose supraorbital nerve and artery exiting the supraorbital foramen and the supratrochlear nerve more medial along the orbital rim. **(c)** Ligation and division of the artery and mobilization of the nerves in preparation for avulsion or division.

never be shaved because the hair may not grow back. After the area is prepared and draped, the skin of the eyebrow is infiltrated with local anesthetic, and an incision is made through the hair of the eyebrow. The incision is slowly and carefully deepened until the branches of the supraorbital and supratrochlear nerves can be identified. The supraorbital nerve usually can be readily identified exiting the supraorbital foramen as a fairly robust bundle composed of several fascicles. The nerve is isolated by using a nerve hook, ensuring that all fascicles have been identified and isolated. The nerve often is accompanied by the supraorbital artery, which can be either spared, if possible, or ligated and divided. By using a fine rongeur to remove a small lip of bone over the supraorbital foramen, a longer segment of nerve can be isolated. Once the nerve has been isolated, it can be removed using a number of different techniques.¹⁸

Classically, a hemostat grasps the nerve, sharply divides it, and then twists to avulse a portion of the nerve from within the foramen. The technique used by the author differs somewhat (**Fig. 13.4**). Once a generous length of the nerve has been exposed, it is placed on stretch and doubly ligated with ligatures of 3-0 silk as far proximal as is technically feasible. The bipolar unit then is used to cauterize the epineurium between the two ligatures, taking care not to violate the epineurium. The nerve then is divided distal to the second ligature and allowed to retract into the foramen. The foramen can then be plugged with bone wax. The supratrochlear nerve also can be removed in a similar fashion through the same incision. This procedure is similar to that used for peripheral neurectomy in the extremities, except that in the extremity the proximal stump is placed into the muscle or a hole that has been drilled in a nearby bone.

Persing and Jane described a slightly different technique, which may be slightly more cosmetic.¹⁹ They place the incision in the supratarsal fold of the upper eyelid, about 9 to 11 mm above the border of the upper eyelash. An incision is made and carried through the orbicularis oris to the level of the orbital septum, which consists of a thin layer of fascia just above the levator oculi muscles. The dissection then is carried out in an avascular plane above the orbital septum to the supraorbital rim, dividing the superior leaf of the corrugator muscle in the process. The supraorbital nerves then are identified and avulsed and the incision closed with an intradermal 6-0 suture.

Infraorbital Neurectomy

Infraorbital neurectomy may be of benefit in patients with trigeminal neuralgia involving the second division and in patients with trigeminal neuropathic pain who have sustained an injury to the nerve during surgery on the maxillary sinus or from periodontal surgery. Infraorbital neurectomy is most effective for pain confined to the cheek and upper lip. It is not nearly as effective for pain involving the roof of the mouth. Pain involving the roof of the mouth and the upper teeth requires an approach to a more proximal portion of the infraorbital nerve or even to the main trunk of the maxillary nerve in the pterygopalatine fossa.

There are several options for infraorbital neurectomy.¹⁸ The infraorbital nerve can be approached extraorally through a skin crease or intraorally. To approach the nerve extraorally, an incision is planned over the infraorbital rim in a skin crease at approximately the junction of the lower eyelid and skin of the cheek. After local anesthetic infiltration, the incision is made and carried deep to the

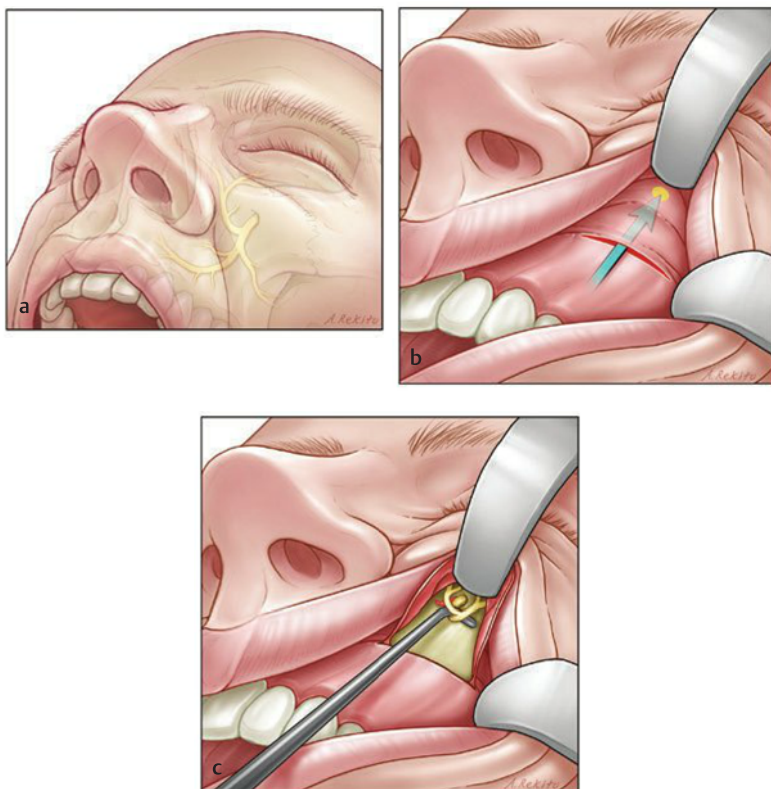


Fig. 13.5 Intraoral exposure of the infraorbital nerve for the purpose of neurectomy via avulsion or ligation/division. **(a)** Position of infraorbital foramen and nerve in relation to surface anatomy. **(b)** Intraoral incision at the gingivolabial margin and dissection trajectory toward infraorbital foramen. **(c)** Infraorbital artery cauterized and infraorbital nerve branches mobilized for avulsion/ligation.

infraorbital rim. The infraorbital nerve is identified exiting its foramen and is removed in the manner described for the supraorbital nerve.

Alternatively, the infraorbital nerve can be approached through an intraoral technique (**Fig. 13.5**). Although this approach is not quite as direct, it has the advantage of not leaving a surgical scar on the face. The patient is positioned supine with the head slightly extended. This should be done carefully in older patients, who often suffer from cervical spondylosis. The upper lip is retracted and the gingivolabial margin is identified. The incision is placed in the gingivolabial margin, beginning with the medial edge starting at the level of the canine tooth and then extending laterally for approximately 2 cm. The incision is deepened to expose the bony maxilla. A periosteal elevator is used to dissect the soft tissues off the maxillary bone superiorly until the

infraorbital nerve is identified emerging from the foramen. Because the anterior wall of the maxillary sinus can be quite thin, caution should be exercised in exposing the bone to avoid entry into the maxillary antrum. The nerve is accompanied by an artery, which should be cauterized and divided to avoid it being torn and retracting into the bone. Because exposing a length of nerve sufficient to ligate may be difficult, the nerve can simply be divided. Then a nerve hook can be inserted into the infraorbital foramen and the stump cauterized. The foramen then is obliterated with bone wax.

Maxillary Neurectomy

In some patients, the distribution of pain may be such that infraorbital neurectomy will not be effective. The maxillary nerve can be accessed using a Caldwell–Luc approach through the maxillary antrum.^{20,21} Once in the maxillary antrum, the posterior wall of the sinus is removed to expose the

Special Consideration

After performing a trigeminal neurectomy, obliteration of the bony foramen from which the nerve had previously exited may enhance the efficacy and duration of pain relief by preventing regrowth of the nerve into the cutaneous and subcutaneous tissues.

periosteum, which covers the retroantral space. The operating microscope then is used for the remainder of the dissection. The first step is identification of the maxillary artery, which is defined and ligated and divided between vascular clips. The infraorbital nerve can be identified running in the roof of the sinus and traced posteriorly to the maxillary nerve. The maxillary nerve then is followed proximally to the foramen rotundum, which represents the safe proximal limit of dissection. The nerve or any of the branches can be ligated and divided between hemoclips, depending on the distribution of the patient's pain. As an alternative to an open procedure, a minimally invasive endoscopic transantral approach also can be performed to gain access to the same anatomical structures.

Inferior Alveolar Neurectomy

Inferior alveolar neurectomy generally is performed for third-division trigeminal neuralgia involving the lower jaw. It is not effective for pain that is located in the tongue. In the latter case, the lingual nerve must be sectioned.²² The inferior alveolar nerve may be approached either intraorally or extraorally.⁷ The extraoral exposure can be performed aseptically and is somewhat easier from a technical standpoint than the intraoral approach. On the other hand, the intraoral approach offers the possibility of simultaneously exposing the lingual nerve in patients who have a significant component of pain involving the tongue.

The extraoral approach is accomplished most easily with the patient under general anesthesia (Fig. 13.6). The patient is placed supine with the head turned to the opposite side. A curvilinear incision is made below and parallel to the angle of the mandible. The incision should be sufficiently below the mandible to avoid injury to the cervical branch of the facial nerve. The skin and subcutaneous tissue are undermined and the masseter muscle identified. The muscle is split in line with its fibers and retracted to expose the lateral surface of the mandible. A bony opening then is made exactly in the center of the mandible using a high-speed drill. Almost immediately after the outer table of bone has been removed, the inferior alveolar nerve will come into view, covered by a layer of

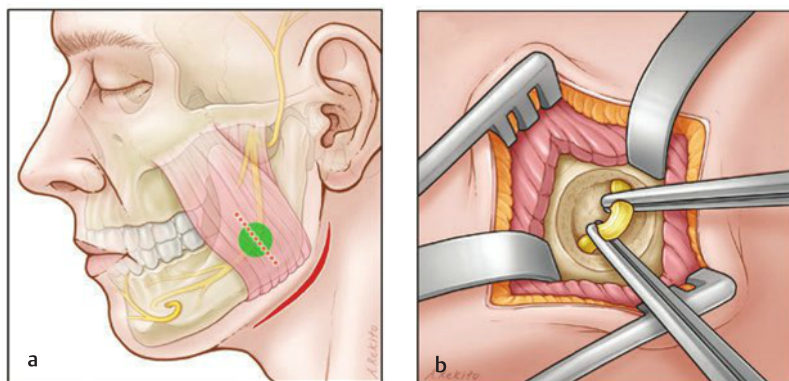


Fig. 13.6 Exposure of the inferior alveolar nerve for the purpose of neurectomy via avulsion or ligation/division. **(a)** Relevant anatomy: skin incision (red curvilinear line), masseter muscle incision (dotted line), and the area of bone removal (green circle). **(b)** Completed exposure with masseter muscle retracted, and inferior alveolar nerve exposed and mobilized in preparation of ligation/avulsion.

fibrous tissue. The nerve is isolated and removed. Hemostasis is obtained and the incision closed in layers in the usual fashion. Inferior alveolar neurectomy can be expected to produce anesthesia of the skin and soft tissues of the lower jaw as well as the lower teeth.

■ Results

It is somewhat difficult to get an accurate handle on the true efficacy of peripheral trigeminal neurectomy. Most of the literature regarding this topic was published in the 1970s and earlier. Indeed, since the early 1990s, articles concerning trigeminal neurectomy have been rare compared with the number that have appeared in the literature concerning microvascular decompression and various percutaneous retrogasserian procedures. Moreover, there have never been any randomized trials regarding the use of neurectomy.

Despite the fact that peripheral neurectomy is a purely palliative procedure, the results would seem to justify its use in carefully selected patients. Indeed, a properly performed peripheral neurectomy almost never fails to produce anesthesia in the desired distribution. It is important to make the patient aware that, despite profound anesthesia, the pain may not resolve completely for several days, and the presence of some immediate residual pain should not be taken as an indication of failure. In most cases, the residual pain is considerably less severe than the pain before the procedure.

In general, most of the larger series that have been published report the average duration of pain relief to be on the order of 2 to 3 years.⁷⁻¹¹ In 1952 Grantham and Segerberg reported on 55 patients who had undergone peripheral neurectomy

and were followed from 6 months to 8 years.¹⁰ The average duration of pain relief in this group was slightly longer than 33 months. Freemont and Miller reported pain relief on the average of about 2 in 26 patients who underwent excision of a total of 43 nerves.²³

Quinn performed a total of 112 neurectomies on 63 patients whose follow-up ranged from several months to 9 years.⁸ The average duration of pain relief ranged between 24 and 32 months. In 1975 Quinn published a supplemental report that included the patients previously reported plus an additional 25 patients.⁹ In all, 162 neurectomies had been performed on 88 patients with trigeminal neuralgia between 1956 and 1971. The operation was successful in providing pain relief in all 88 patients; however, slightly more than 50% (48 of 88) of the patients experienced some immediate residual pain, which persisted for an average of about 6 days (range, 1 to 21 days) before resolving. The median pain-free period among the 88 patients was 41 months (mean, 52 months). There was no significant difference in the pain-free intervals when patients with mental (37.5 months), inferior alveolar (38 months), and infraorbital (38.5 months) neurectomies were compared; however, lingual neurectomy provided a slightly longer pain-free interval (44 months), which Quinn postulated was related to the slower regeneration of nerve lying completely within soft tissue.

Mason reported the results of 47 neurectomies performed in 36 patients, including 32 infraorbital and 15 inferior alveolar neurectomies.⁷ Twenty-one infraorbital and all the inferior alveolar neurectomies were carried out as primary procedures; 11 patients underwent infraorbital neurectomy for at least the second time. Mason defined failure as the point where further medical or surgical treatment was required to achieve pain control. The failure rate at the end of 1 year was 36% and at the end of 4 years, 74%. There was no difference in the pain-free incidence between those patients undergoing primary and secondary procedures. In general, infraorbital neurectomy was more effective than inferior alveolar neurectomy. Mason noted that in patients who underwent infraorbital neurectomy, failure to occlude the bony foramen resulted in a statistically higher failure rate.

More recently, Murali and Rovit examined the efficacy of trigeminal neurectomy performed in 40 patients.¹¹ They performed a total of 69 neurectomies, including 28 on the supraorbital and supratrochlear nerves, 40 on the infraorbital nerve, and a single inferior alveolar procedure. The series included 28 patients who had previously undergone trigeminal ganglion radiofrequency thermocoagulation 6 weeks to 5 months before the neurectomy. The results were stratified in terms of pain relief into three categories: *excellent* (total pain relief without the need for pharmacological therapy, e.g., carbamazepam); *good* (residual pain requiring "modest" amounts of carbamazepam); and *poor* (no significant pain relief despite adjunctive treatment with carbamazepam). The best outcomes occurred in the group who had previously had a radiofrequency gangliolysis. Twenty-two patients (79%) in this group were judged as having excellent pain relief, which persisted in excess of 5 years; the other 6 patients had a good result. In the group who underwent neurectomy as a primary procedure, 7 (58%) had excellent pain relief and 5 (42%) were classified as good. Six (15%) of the 40 patients experienced pain recurrence after a mean period of 24 months and were treated with an additional neurectomy. All these patients had complete pain relief with an average follow-up of 2 years.

■ Conclusion

Although peripheral trigeminal branch neurectomy has largely been supplanted by other surgical procedures that provide better long-term pain relief, it can still be considered a valuable option in selected patients. Peripheral neurectomy is especially useful in treating older debilitated patients who suffer from trigeminal neuralgia and who cannot undergo a more substantive procedure. It also may offer pain relief to persons with trigeminal neuropathic pain and even highly selected patients with atypical facial pain. The procedures are relatively easy to perform and are associated with low morbidity. However, as is always the case in treating patients with facial pain disorders, it is imperative to establish the correct diagnosis right from the outset to make good clinical decisions.

Nonetheless, in spite of all the newer procedures currently available, peripheral branch trigeminal neurectomy can still be a valuable modality of treatment in the overall armamentarium of the surgeon who is involved in treating patients with chronic refractory facial pain syndromes.

Editor's Comments

Dr. Osenbach has provided an excellent review of the peripheral anatomy of the trigeminal nerve, and established techniques for trigeminal nerve block and neurectomy.

I would have to disagree with his opening statement:

These particular techniques are currently more of historical than practical interest, given the alternatives such as the minimally invasive percutaneous needle techniques (radiofrequency gangliolysis, glycerol rhizolysis, and balloon microcompression) and stereotactic radiosurgery, especially for the treatment of trigeminal neuralgia.

What he has demonstrated is that these procedures are low morbidity and relatively straightforward to accomplish, and that they should be preserved in the surgical armamentarium to combat facial pain. He presents two case series with excellent results in 79 to 100% of patients from 41 months to in excess of 5 years. These outcomes compare very favorably to those of both trigeminal radiofrequency gangliolysis and radiosurgery.

Pain surgery, like all of medicine, can sometimes be needlessly "trendy." Older techniques, with demonstrated efficacy, fall out of favor and are sometimes lost to future generations of surgeons simply because the practices are not passed along. I would hope that this chapter, and the continuing attention of surgeons who deal with facial pain, will promote the preservation of a wide spectrum of surgical options for facial pain.

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Stereotactic Radiosurgery for Trigeminal Neuralgia

Douglas Kondziolka and L. Dade Lunsford

Trigeminal neuralgia is described by patients as one of the most severe pain disorders. When medical management fails to control the pain of trigeminal neuralgia, patients require surgical intervention. Effective surgical procedures include craniotomy and microvascular decompression, percutaneous ablative procedures,¹ and stereotactic radiosurgery. All surgical procedures have variable but definite rates of risk and pain recurrence.

Gamma Knife (Elekta, Stockholm, Sweden) stereotactic radiosurgery (GKSr) is a minimally invasive surgical approach for managing trigeminal neuralgia. In 1951 Lars Leksell advocated radiosurgery for trigeminal neuralgia using a prototype guiding device linked to a dental X-ray machine.^{2,3} During the next 50 years, new radiosurgery devices were developed and used for this indication.⁴⁻⁹ This report evaluates the effectiveness of GKSr for pain relief, the time until relief occurs, the durability of pain relief, and clinical factors associated with success or complications. When the current technique of trigeminal neuralgia radiosurgery that targets the nerve just anterior to the pons using high-resolution magnetic resonance began in 1992, it was not long before the concept was widely adopted. A literature search (3/8/2013) noted 117 articles under “trigeminal neuralgia, gamma knife” (www.world-sci.com). Other radiosurgery techniques using modified linear accelerators have also been used for trigeminal neuralgia.

■ Considering Gamma Knife Radiosurgery versus Other Surgical Options

The role of GKSr in the management of medically refractory trigeminal neuralgia has evolved. It is important to place this surgery in the context of other therapeutic modalities for this disorder.¹⁰ By the end of the 20th century, craniotomy and microvascular decompression had emerged as the “gold standard” surgical interventions for medically refractory trigeminal neuralgia in eligible patients; at 10 years 64% of patients had complete relief (defined as the absence of lancinating facial pain, or a reduction in pain of at least 98% off medication—Barrow Neurological Institute, or BNI, outcome grades I and II). Nine percent noted partial relief (defined as a 75% reduction in pain with or without medication [BNI grade IIIb]).¹¹ Unfortunately, many patients with trigeminal neuralgia are unsatisfactory craniotomy candidates because of the associated risks of advanced age or the presence of medical comorbidities.¹² Stereotactic radiosurgery is the least invasive modality for such patients.

There are different ways to report pain relief. These include outcomes that separate relief into complete (no pain), partial (significantly improved but occasional

or less severe pain), and inadequate (failure). In addition, whether or not patients can reduce or eliminate medical therapy is important. The BNI score is one such approach that includes these different outcomes: Grade I is complete relief off all medication; grade II is partial relief off medication; grade IIIa is complete relief with medication; and grade IIIb is partial relief with medication.¹³ These four groups constitute “successful” pain relief, although some consider only outcome grades I to IIIa as successful. BNI scores IV and V are usually considered treatment failures because they represent limited or even no pain relief.

The goal of trigeminal neuralgia (TN) surgery is complete elimination of pain and the need for medication. Achieving this entails a balance between surgical risk, maintenance of trigeminal nerve function, and acceptable rates of pain relief. Not all procedures relieve pain and not all patients may be able to eliminate medication. For patients who have failed one or more surgical procedures, the expectation of complete relief by additional procedures is reduced. For many patients, pain reduction may be an acceptable outcome, particularly if medication-related side effects are reduced and performance of daily activities is improved and more comfortable. In addition, in view of the vagaries of pain assessment and the periodic nature of pain associated with trigeminal neuralgia, a patient may report having no pain at one assessment (grade I), only to have it recur at various levels in the future. There is a need to report not only pain outcomes but outcomes that describe the effect of pain on daily living and quality of life.

It is important to compare stereotactic radiosurgery with other minimally invasive surgical strategies such as percutaneous rhizotomy. Kanpolat et al¹⁵ reported on 1,600 TN patients who underwent RFL. At 5-year mean follow-up, 58% were pain free but 42% had partial or complete recurrence. At an average of 20 years, the pain-free rate decreased to 41%. Other studies showed that the pain-free rate following RFL varied from 20 to 82% and recurrence or failure varied from 20 to 80%.^{15–19} With RFL, an increased rate of facial sensory dysfunction has been correlated with longer pain relief, but also with various degrees of sensory dysesthesias. Tatli et al²⁰ reported a meta-analysis including microvascular decompression (MVD) and RFL from a total of 28 studies. They concluded that MVD had superior outcomes compared with RFL. Although RFL provided a high rate of initial pain relief, the average pain-free rate was 50.4% at mean follow-up of 5 years.

■ Gamma Knife Radiosurgery Technique for Trigeminal Neuralgia

Patients with trigeminal neuralgia are evaluated with high-resolution magnetic resonance imaging (MRI); computed tomography (CT) may be substituted in patients who cannot undergo MRI scans. Prior imaging is important to rule out a compressive tumor or vascular malformation. Gamma Knife radiosurgery begins with rigid fixation of an MRI-compatible Leksell stereotactic frame (model G, Elekta) to the patient's head. Local anesthetic scalp infiltration (5% Marcaine and 1% Xylocaine) is used, supplemented by mild intravenous sedation as needed. High-resolution images are acquired with a fiducial system attached to the stereotactic frame. For trigeminal neuralgia radiosurgery, a three-dimensional (3D) volume acquisition MRI using a gradient pulse

sequence (divided into 28 to 36 1-mm-thick axial slices) is performed to cover the entire region and surrounding critical structures. A T2-weighted 3D volume sequence is performed to visualize the cranial nerves and can be helpful in certain patients, particularly after prior microvascular decompression. The entire cranial vault is assessed with a 3-mm T2 study to exclude other, unrelated pathologies in addition to providing a volumetric rendering of the head for dose planning. Planning is performed on narrow-slice-thickness axial MR images with coronal and sagittal reconstructions. The sagittal view provides excellent identification of the course of the nerve in that plane.

■ Radiosurgical Dose Planning

Dose planning is a critical aspect of radiosurgery, and image-integrated software (GammaPlan, Elekta) provides the platform for reliable and accurate nerve irradiation. Specific Gamma Knife radiosurgery techniques include accurate definition of the nerve anterior to the pons using small collimation (one or occasionally two 4-mm isocenters)¹⁴ (Fig. 14.1).

After optimizing the plan, a maximum dose to the nerve target is determined. The treatment isodose, maximum dose, and dose received by any ad-

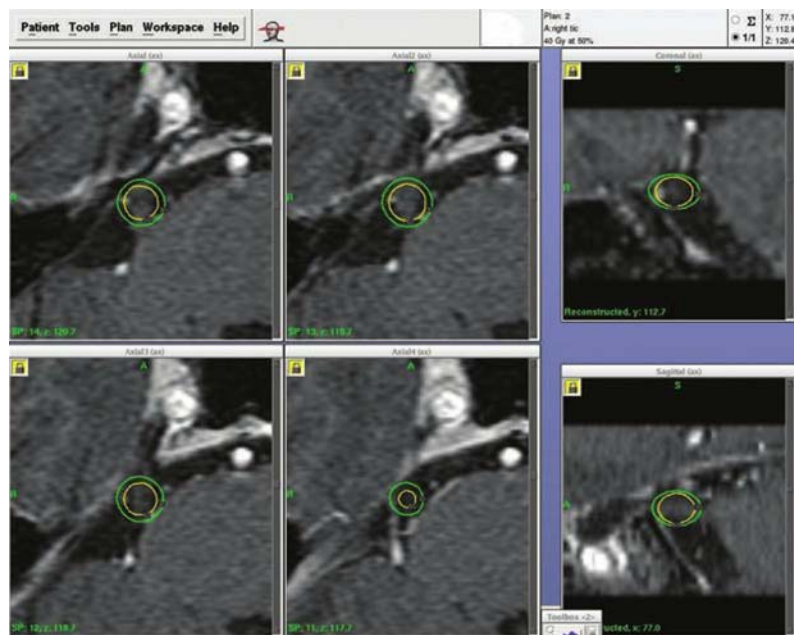


Fig. 14.1 Trigeminal neuralgia radiosurgery dose planning. A single 4-mm isocenter is targeted at the nerve. The 30% isodose line is at the brainstem surface. The sagittal image (*lower right*) shows the horizontal course of the nerve.

jacent structures are jointly selected by a neurosurgeon, radiation oncologist, and medical physicist. In Gamma Knife radiosurgery a dose of 80 to 90 Gy is typically prescribed by most centers to the 100% (maximum) isodose line. The dose fall-off at the 20 or 30% line is also examined. It is prudent to keep the brainstem dose low, but this is achieved with small beam collimation without beam channel or sector blocking. Dose prescriptions for trigeminal neuralgia evolved in the mid-1990s (with a range of 60 to 90 Gy) and now are consistent at most centers. A maximum dose of 80 Gy is associated with a low rate of facial sensory dysfunction (about 10%) with an approximate 80% success rate for significant pain relief. Some centers have used 85 or 90 Gy as the maximum dose to increase the radiobiologic effect. Clinical trials that compared one- and two-isocenter radiosurgery (where a longer segment of nerve would be irradiated) were performed. These showed that the rate of sensory dysfunction increased but that pain relief was not significantly affected.²¹

After radiosurgery, patients are followed up with serial clinical assessments that are commonly requested at 3 months and then annually or as needed. If an MRI with contrast is obtained within the first 2 years, contrast enhancement within the nerve target site is commonly seen (**Fig. 14.2**).

Other centers, using modified linear accelerators, have reported clinical results in relatively small patient case volumes. Frame-based stereotactic radiosurgery (SRS) facilitates accurate delivery of dose to the target while sparing adjacent pontine and temporal lobe structures. Subsequent experience with linac-based radiosurgery has provided additional concern. A commercially available product that links two technologies led to radiation injury to the brainstem and concom-

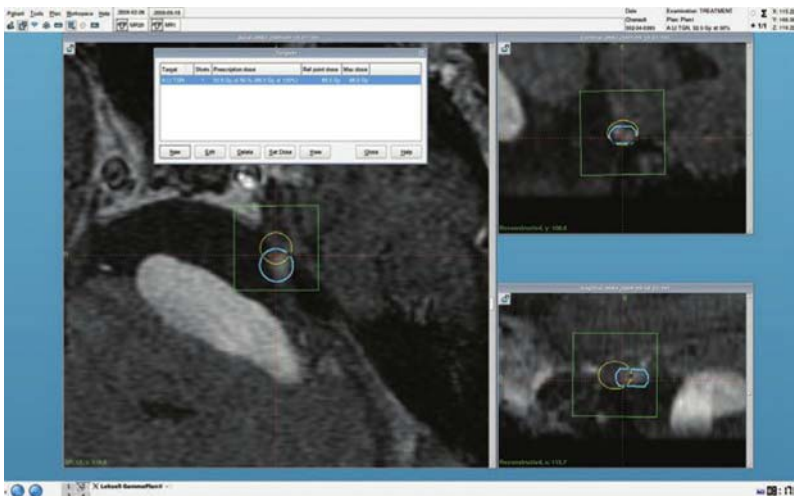


Fig. 14.2 Repeat trigeminal neuralgia radiosurgery. The original dose plan on the proximal nerve shows contrast enhancement at that target. The first dose was 80 Gy and the second, 65 Gy. The patient had pain from dolichoectatic vascular trigeminal nerve compression.

itant neurological deficits (*New York Times*, 12/28/2010; http://www.nytimes.com/2010/12/29/health/29radiation.html?_r=0). A recent report from Italy described 23 patients who underwent linac (i.e., linear accelerator) radiosurgery (margin dose of 40 Gy) and compared outcomes with 22 patients who underwent hypofractionated radiation (fractionated total dose of 72 Gy) using a mask and bite block immobilization system.²² The authors reported that patients obtained better pain relief after radiosurgery. Radiosurgery is designed to obtain a limited radiobiologic effect that includes nerve demyelination at a specific site and via a specific volume. Fractionation provides no radiobiologic advantage in lesion generation, but for technologies that have poor dose fall-off beyond the target volume (poor selectivity), it may provide less risk of collateral damage. Quality radiosurgery is dependent on reliable technology, rigid target immobilization, high-resolution imaging, proper dose selection, and delivery in a single treatment session.

■ Gamma Knife Radiosurgery: Clinical Results

Achievement of Pain Relief

Most centers report an average latency to pain relief after radiosurgery of approximately 1 to 2 months.^{14,23–26} In our own study we found that 89% of patients responded to treatment at a median of 1 month.²⁷ We found that patients with typical trigeminal neuralgia, patients who underwent GKSR as their initial surgical procedure, and patients who underwent earlier GKSR (< 3 years) after pain onset had faster pain relief (grades I–IIIb). The median time to achieve complete pain relief (grade I) was 5 months. By 12 months after GKSR, 11% of patients still had pain. Patients who continued to suffer disabling pain required an additional surgical procedure. We advocate repeat GKSR only if complete pain relief had been achieved initially, with subsequent recurrence.²⁵ A recent analysis by Park et al is the largest series of repeat Gamma Knife radiosurgery with outcomes similar to a first procedure.²⁸ Typically, results are not as good as after a first procedure, with fewer patients reaching complete pain relief. Still, many patients are improved after repeat radiosurgery (**Fig. 14.2**).

In a large recent series from Wake Forest University with 777 Gamma Knife radiosurgery procedures performed between 1999 and 2008 (448 evaluable patients), the authors found that 83% of patients achieved pain grades of I to III, with 43% achieving grade I.⁶ Twenty-six percent developed some postradiosurgery facial numbness, which they correlated with the nerve entry zone dose; however, their mean dose was 88 Gy. Increasing sensory loss correlated with pain relief.

Maintenance of Pain Relief

Our experience indicates that the majority of patients experience lasting, satisfactory pain reduction with few complications after GKSR. In our series, 75% of patients achieved or maintained pain control (BNI grades I–IIIb); 59% had pain relief at 3 years; 43% maintained relief at 5 years; and 29% were still controlled with or without medications at 10 years.²⁷ We found that younger patients (< 65 years),

those with atypical pain features, and those who had undergone more than three prior surgical procedures were more likely to suffer earlier pain recurrence. The best result, BNI grade I (pain free, off medication), was achieved in 28% of patients. Of those with a significant pain recurrence, 60% underwent further surgery and 40% were maintained on medication alone. These findings are consistent with the results reported by other centers.^{13,27,29–32}

Villavicencio et al reported that 95 TN patients who underwent CyberKnife (Accuray, Sunnyvale, CA, USA) radiosurgery had an initial pain relief rate of 67%. Fifty percent maintained complete pain relief, defined as > 90% pain relief without medication (BNI grades I and II), but 47% developed postradiosurgery sensory loss. Sensory loss did correlate with better pain relief.³³ In addition this report noted that 18% of patients developed other side effects such as masticator weakness, diplopia, and decreased hearing. Such avoidable complications are related to excessive dose delivery to adjacent structures, poor target definition (often based on CT rather than MRI nerve definition), or selection of the trigeminal ganglion as the target. The median length of nerve irradiated was 6 mm (range, 5–12 mm), which is longer than the typical GKSR target length using a single isocenter. In our previous prospective randomized study for TN patients who underwent GKSR using one or two isocenters, longer nerve irradiation resulted in a higher complication rate with no difference in efficacy.²¹ The common Gamma Knife radiosurgery technique utilizes one isocenter and a limited target nerve segment that lies between the ganglion and the pons.

Sensory Dysfunction after Gamma Knife Radiosurgery

Postrhizotomy paresthesias or sensory loss of varying degrees is observed in 6 to 70% of patients after thermal rhizotomy, glycerol rhizotomy, or balloon microcompression, depending on the technique.^{22,23,25,29,30,34–36} Some investigators believe that the onset of sensory loss is associated with longer term pain relief. In our report, sensory dysfunction was found in 11% of patients, a lower incidence than noted in the Wake Forest series, perhaps related to the average higher dose used at that center.⁶ Only one patient in our experience (0.19%) has developed deafferentation pain. Ten of 53 patients who later developed facial sensory dysfunction reported pain recurrence after GKSR. The 1-, 3-, and 5-year rates for maintenance of pain relief in patients who later developed facial sensory symptoms were 91%, 82%, and 78%, respectively. These data suggest that patients who developed facial sensory symptoms had a reduced rate of recurrent pain ($p < 0.0001$). Pollock et al reported a significant association between higher radiation dose and increased risk of trigeminal neuropathy; 45% of the patients had received a maximum dose of 90 Gy and reported a trigeminal deficit as opposed to 15% who received less.^{37,38} They reported a lower rate of pain recurrence in patients with sensory symptoms (15% versus 41% in those without), but this did not reach significance ($p = 0.08$).

Consistent with our own findings, Brisman reported a 5% complication rate in patients irradiated to doses between 70 and 80 Gy.^{29,30} Henson et al reported that 54% of patients who underwent percutaneous retrogasserian glycerol rhizotomy (PRGR) and 30% of patients who underwent GKSR later developed facial sensory symptoms. A higher morbidity rate was found after PRGR ($p = 0.018$).³⁹

Comparison of Gamma Knife Radiosurgery with Other Surgical Approaches: Level 2 Studies

The outcomes of Gamma Knife radiosurgery can be compared with those from other surgical options (Table 14.1). There has not been a randomized controlled trial to compare radiosurgery with any other treatment.

Henson et al³⁹ reported on a series of 36 patients who underwent PRGR and 63 patients who underwent GKSR for trigeminal neuralgia. They found that 86% of patients who underwent PRGR and 92% of patients who underwent GKSR achieved a successful treatment outcome, defined as BNI grades I to IIIb ($p = 0.49$). Fifty-three percent of patients who underwent PRGR and 41% of patients who underwent GKSR experienced pain recurrence or no pain relief at median recurrence times of 5 and 8 months, respectively ($p = 0.30$). Although PRGR worked faster on average, GKSR lasted longer. Thus, radiosurgery appears to have a lower rate of pain relief in longer term follow-up (5–10 years), but it also has the lowest morbidity or associated sensory dysfunction. To date no longitudinal assessment of quality of life exists after any of the surgical procedures for TN. Most agree that the three key indicators of such quality are pain relief, new sensory symptoms, and medication tolerance if used.

Pollock and Schoeberl reported results from 91 patients who underwent MVD and 49 who had GKSR.⁷ The patients having MVD were younger (mean, 58 vs. 67 years; $p < 0.001$). Although both groups did well, those who underwent MVD were pain free off medication at a higher rate (84% vs. 66% at 1 year; 77% vs. 56% at 4 years). Complications after MVD included cerebrospinal fluid leakage ($n = 3$), hearing loss ($n = 2$), wound infection ($n = 1$), pneumonia ($n = 1$), and deep vein thrombosis ($n = 1$). The only morbidity after GKSR was facial sensory dysfunction ($n = 20$), which was also seen after MVD ($n = 19$). Thus, MVD had a higher rate of complete pain relief but more general morbidity and was performed in younger patients.

In a similar study to compare GKSR and MVD, Brisman reported 61 patients after GKSR and 24 after MVD.³⁰ No significant difference was found between pain outcomes. After MVD, 68% were pain free, and the result was 59% after GKSR ($p = 0.09$). For pain improvement, the results were 90 and 75%, respectively ($p = 0.17$).

In summary, radiosurgical pain relief results appear similar to the results reported by patients who undergo initial microvascular decompression.^{30,40} Microvascular decompression is designed to obtain relief by solving one particular

Table 14.1 Studies comparing gamma knife (GK) radiosurgery with other surgeries

Author	Number of patients: GK	Number of patients: other*	Improved (%): GK	Improved (%): other	Other surgery
Henson ³⁹	63	36	92	86 (BNI I–IIIb)	Glycerol rhizotomy
Pollock ⁷	49	91	77	84 (BNI I)	MVD
Brisman ³⁰	61	24	58	68 (BNI I)	MVD
			75	90 (BNI I–IIIb)	

*Glycerol rhizotomy or microvascular decompression (MVD).

etiology of pain for many patients: vascular cross-compression of the root entry zone of the trigeminal nerve. We continue to advocate craniotomy and microvascular decompression for younger patients suitable for invasive surgery. The benefit of decompression is less if pain recurrence requires a second MVD. GKSR as an alternative minimally invasive procedure designed to reduce patient risk is clearly well tolerated, has a strong safety profile, and pain outcomes that are consistent between Gamma Knife centers.

Editor's Comments

Few question whether stereotactic radiosurgery (SRS) has a role in the management of medically intractable trigeminal neuralgia (TN). The only serious debate is over the question of the outcome from SRS, and how that compares with other interventions. Part of this discussion is how to define pain relief after interventions for TN.

The Barrow Neurological Institute (BNI) scale defines six grades of facial pain relief: I, no pain, no medications; II, occasional pain, no medications required; IIIa, no pain, medication; IIIb, pain, medication controlled; IV, pain, not well controlled; and V, no pain relief. Drs. Kondziolka and Lunsford mention that "some consider only outcome grades I to IIIa as successful." For the record, I count myself as one of those "some."

I submit that the goal of surgery for TN is pain relief. To label "pain, medication controlled (but not gone)" as successful is generous at best. In fact, Dr. Kondziolka has tacitly acknowledged this in an article in which he refers to "significant" pain relief. In that series²⁷ significant pain relief (BNI grades I–IIIa) after GKSR (gamma knife stereotactic radiosurgery) was achieved in 73% at 1 year, 65% at 2 years, and 41% at 5 years. In that series, which included grade IIIb, a BNI grade of I to IIIb was found in 80% at 1 year, 71% at 3 years, 46% at 5 years, and 30% at 10 years—a substantial difference. From these data I would estimate that the median pain-free survival after SRS is between 3 and 4 years.

Direct comparisons of SRS and microvascular decompression (MVD) have been conducted. Linskey and colleagues⁴¹ looked prospectively at a nonrandomized cohort of patients with TN who underwent either MVD or SRS. Initial and follow-up pain-free rates were 100% and 80.6% for MVD and 77.3% and 45.5% for GKSR. The median time to the maximal benefit after GKSR was 4 weeks (range, 1 week–6 months). The initial, 2-, and 5-year actuarial pain-free rates were 100%, 88%, and 80% for MVD and 78%, 50%, and 33% for GKSR ($p = 0.0002$). The relative risk of losing pain-free status by 5 years post-treatment was 3.35 for patients in the GKSR group compared with the MVD group. The respective rates of permanent mild and severe sensory loss were 5.6% and 0% for patients in the MVD group, and 6.8% and 2.3% for patients in the GKSR group. In this series, MVD was distinctly superior to SRS for the management of TN.

SRS is an important strategy for managing TN. It is a destructive procedure, and it is a form of noninvasive trigeminal rhizolysis. In terms of outcome assessment, it probably compares most favorably with radiofrequency rhizolysis. MVD is still the "gold standard," but another destructive procedure, posterior sensory rhizotomy (PSR), must be better studied. Ultimately, excellent and durable outcomes should determine which procedures should be used on patients with TN, in whom medication has failed to relieve their pain. I respect the pioneering efforts of Drs. Kondziolka and Lunsford in this innovative therapy, and the integrity required to report their results in a rigorous manner.

Conclusion

Gamma Knife radiosurgery has become a well-documented management option for patients with trigeminal neuralgia. It is both safe and effective and is the least invasive surgical option. Reports from centers worldwide show consistent

outcomes, and longer term data are now available from numerous centers. Outcomes data are consistent because methods of targeting and radiosurgical delivery have been consistent.

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Percutaneous Radiofrequency Trigeminal Gangliolysis for Trigeminal Neuralgia

Kim J. Burchiel

Denervation of the receptive fields of the triggering zones has long been known to be a successful strategy to control the pains of trigeminal neuralgia (TN). Initial procedures involved either peripheral surgical neurectomy or neurolytic injection into relevant trigeminal branches.¹ In the 19th century it was also recognized that subtemporal trigeminal rhizotomy, or ganglionectomy, could produce long-lasting pain relief.^{2–4} In fact, Cushing was one of the first innovators of the subtemporal approach.⁵

The first electrocoagulations of the trigeminal ganglion using radiofrequency (RF) diathermy were performed by Kirschner in 1932.⁶ These lesions were made with a large (1-cm uninsulated tip) electrode. Damage to adjacent cranial nerves from the procedure, and the associated neurologic deficits, limited the usefulness of this approach. Percutaneous gangliolysis did not advance until White and Sweet⁷ refined the RF technique by: (1) the use of short-acting anesthetic agents to produce brief and intermittent analgesia, immobility, and amnesia; (2) electrical stimulation for localization of the electrode within target roots; (3) a reliable RF generator; and (4) a thermistor within the RF electrode to monitor temperature during the lesion process. Following these improvements, the RF technique became the procedure against which subsequent percutaneous techniques for TN were compared.

The complications of trigeminal deafferentation continue to challenge the practice of RF gangliolysis. Advances in electrode and RF generator technology have helped to minimize the dreaded outcome of anesthesia dolorosa (or its lesser variants) and “neuroparalytic keratitis.” Nevertheless, much of the morbidity associated with this procedure is dependent on the sensory loss objective, and experience, of the neurosurgeon. Despite early suggestive evidence, damage to the trigeminal rootlets by RF heating does not appear to be selective to smaller pain-conducting axons (C and Ad fibers).⁸ Damage to trigeminal afferents appears to be a *quantitative* phenomenon, but preservation of touch sensation, and concurrent hypalgesia, can be achieved by carefully graded lesions with contemporaneous testing of the patient’s facial sensation during the procedure.

■ Principles

Trigeminal rhizotomy, by any form, stands out in the treatment of a neuropathic pain in that sensory loss, up to a critical point, reliably stops the characteristic and “classic” triggerable lancinating, stabbing, or shocklike pains of type 1 TN (TN1).

For almost every other neuropathic pain syndrome, the production of sensory loss is ineffective in alleviating the pain, and has a substantial chance of making it worse. Arguably, TN1 is not a “typical” neuropathic pain,⁹ which is typified by a constant burning or otherwise unpleasant sensation. It is the brief nature of trigeminal neuralgia and the typical sensory triggers that set it apart. Most probably, it is the consequential blunting of the triggering input that allows remission from TN after rhizolysis or gangliolysis that is the basis for its efficacy.

Devor et al have proposed the most coherent physiological theory of TN.¹⁰ They hypothesized that a triggering stimulus, touch or proprioceptive, within large trigeminal afferent fibers sets off an abnormal discharge in the retrogasserian root. This discharge, in turn, antidromically invades the gasserian ganglion, thereby “igniting” a wave of depolarization within the ganglionic cell bodies produced by a chain reaction of incremental extracellular release of excitatory neurotransmitters. Direct human observation by microelectrode recordings supports this proposed mechanism.¹¹

Therefore, as our neurosurgical predecessors consistently advised, denervation of the trigger areas of TN should be the goal of these destructive procedures. If this can be accomplished, then triggering stimuli will fail to produce the abnormal retrogasserian after-discharge, and no ignition will occur in the ganglion. The majority of trigeminal afferents terminate in the perioral area, so it should be no surprise that most trigger areas surround the mouth, and that most denervating procedures, including percutaneous radiofrequency trigeminal gangliolysis (PRTG), target the mandibular (V3) and maxillary (V2) divisions.

■ Practice

Patient Positioning

The patient is brought to the operating room and an appropriately sized nasopharyngeal airway is selected based on the patient's nasal passages.

The patient is positioned to obtain an image of the foramen ovale, by either the submental vertex or oblique fluoroscopic projection, and the entry point on the face is sterilely prepared and draped. After an initial amnestic dose of propofol is given, the nasopharyngeal airway, which has been lubricated with lidocaine jelly, is inserted into the nostril. Once this is accomplished, a second bolus of propofol is then administered to induce brief general anesthesia. Respiration can be supported through the nasopharyngeal tube, if necessary, by combining occlusion of the opposite nares with forward displacement of the mandible (“jaw thrust”), using the anesthesia circuit. If the patient is made briefly apneic by the propofol bolus, respirations will promptly resume after a few breaths are administered via the nasopharyngeal tube.

Placement of the Electrode

Once the patient is surgically anesthetized as evidenced by the loss of the eyelash reflex, a no. 11 blade is used to make a stab incision approximately 2.5 cm lateral to and 1 cm inferior to the labial commissure (**Fig. 15.1**). The TEW cannula with the stylet is then inserted toward the foramen ovale, with one finger placed with-

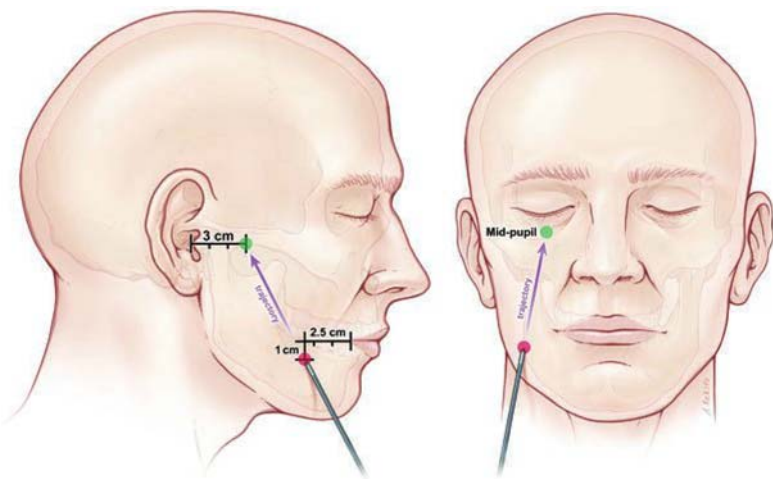


Fig. 15.1 Once the patient is surgically anesthetized a no. 11 blade is used to make a stab incision approximately 2.5 cm lateral to and 1 cm inferior to the labial commissure.

in the oral cavity to prevent violation of the oral mucosa. The line of insertion is the junction of two planes from the insertion site: to a point 3.0 cm anterior to the ipsilateral tragus, and to a point at the medial border of the ipsilateral pupil (Hartle technique) (**Fig. 15.2**). The cannula is advanced until it contacts the skull base, radiographically anterior to, and in line with, the foramen ovale. The cannula is then gradually redirected posteriorly until the foramen is acquired. Commonly, entering the foramen ovale is accompanied by a jaw jerk.

Once the foramen ovale is entered, the fluoroscope is moved into the lateral position, and the cannula is advanced until an appropriate depth is reached. For V1 or V2 trigeminal neuralgia, a curved electrode is often needed. Generally, the curved electrode will not need to be positioned beyond the clival line on lateral fluoroscopy. For V3 trigeminal neuralgia, a straight electrode can be used, and the cannula is positioned well anterior to the clival line. The trigeminal cistern lies just anterior to the nexus of the clivus and the petrous bone on lateral fluoroscopy. Positioning of the electrode tip such that it points more toward the posterior clinoid allows acquisition of the more medial V1–2 divisions, as deeper advancement of the electrode transits this part of the retrogasserian roots (**Figs. 15.3** and **15.4**). **Fig. 15.5** shows the sequential appearance of the procedure on lateral fluoroscopic imaging from electrode penetration of the foramen ovale (**Fig. 15.5a**), followed by straight electrode (**Fig. 15.5b**) and curved electrode (**Fig. 15.5c**) placement.

Stimulation and Lesion Generation

Once the radiofrequency electrode is in a radiographically satisfactory position, the patient is allowed to awaken. Stimulation testing is then performed (rate of 50 Hz and a pulse width of 1 ms) with the awake and cooperative (but still sedated and usually amnesic) patient to ascertain that the desired divisions

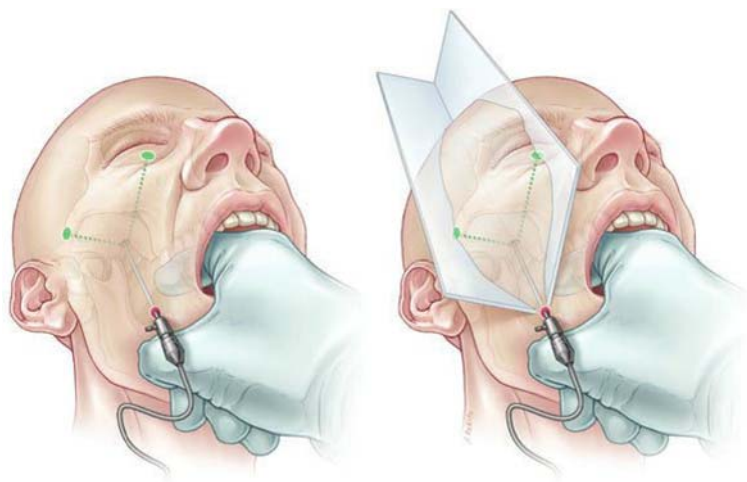


Fig. 15.2 The cannula with the stylet is inserted toward the foramen ovale, with one finger placed within the oral cavity to prevent violation of the oral mucosa. The line of insertion is the junction of two planes from the insertion site: to a point 3.0 cm anterior to the ipsilateral tragus, and to a point at the medial border of the ipsilateral pupil.

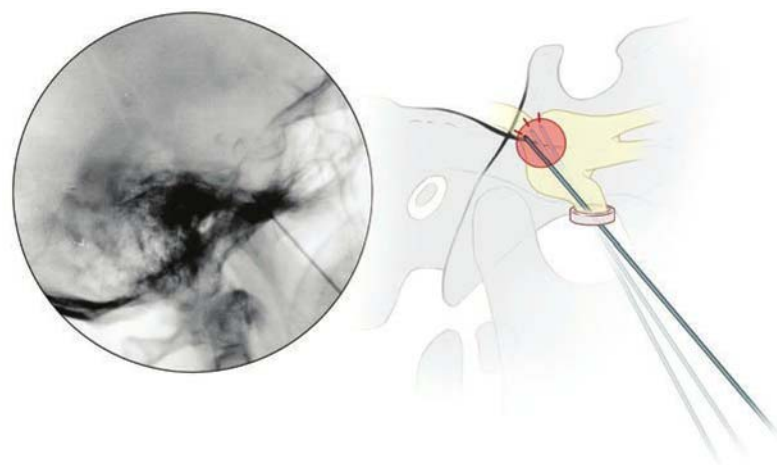


Fig. 15.3 Once the foramen ovale is acquired on the submental vertex or oblique view of the skull base, the fluoroscope is moved into the lateral position, aligning the internal auditory canals to attain a true lateral view. The cannula may then be advanced, first through V3, then sequentially through V2 and V1. Deeper penetration is needed to reach V2 and V1, and a more superior trajectory is more likely to achieve access to V1. The retrogasserian cistern (red circle) is generally located just anterior to the nexus of the petrous bone and clivus on lateral fluoroscopy.

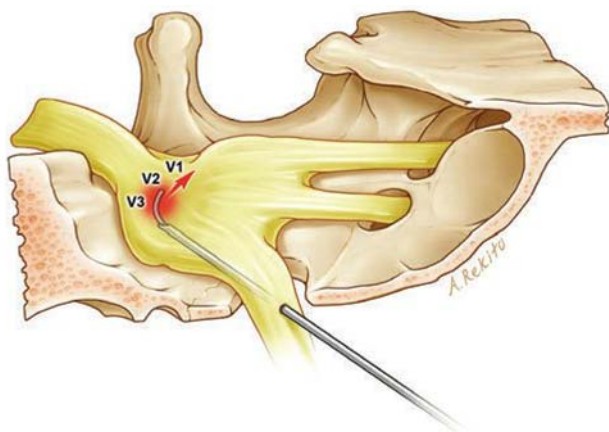


Fig. 15.4 As the cannula penetrates the ganglion from lateral to medial, it should pass from V3 to V2, and eventually to V1. Based on the angle of attack, and the geometry of the patient's petrous bone and ganglion, a straight cannula can pass from V2 directly through the dura propria and become subtemporal. A curved electrode, introduced into V3, can help to reduce this possibility.

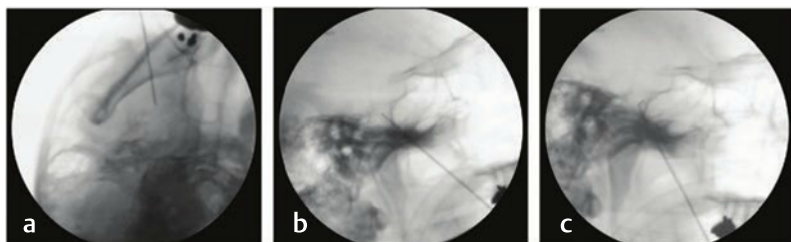


Fig. 15.5 (a) Submental vertex fluoroscopic view showing acquisition of the foramen ovale by the cannula. When the cannula is first inserted, a “safe zone” anterior to the foramen, on a trajectory to intercept it, is first encountered, and then the electrode is gradually directed posteriorly under fluoroscopic control until the foramen is entered, typically accompanied by a jaw jerk. (b) Lateral fluoroscopic view showing the straight electrode in a position at which stimulation should produce V3, and possibly V2, paresthesias. (c) Lateral fluoroscopic view showing the curved electrode in a position at which stimulation would likely produce V2 and possibly V1 paresthesias.

of the trigeminal nerve are being targeted. Once this has been accomplished, another bolus of propofol is given for sedation and to allow the creation of the first lesion. For V2 and V3 lesions, the first lesion is often made at 75 to 80°C for 90 seconds. For V1 trigeminal neuralgia, a lesion at 70 to 75°C may be advisable to minimize the risk of corneal anesthesia.

Once the lesion has been made, the patient is again awakened and several minutes are allowed to pass such that the patient demonstrates reliable and

consistent responses to testing. A facial sensory examination is performed, with the patient asked to discriminate between pin prick and light touch sensation. The goal of the lesion is the loss of the patient's ability to differentiate sharp and dull sensation. Stimulation testing (50 Hz at 1 ms pulse duration) can again be performed to ensure that the RF electrode is targeting the desired division of the trigeminal nerve. Once this is confirmed, the patient is resedated with a bolus of propofol, and further cycles of lesion production can be completed, raising the lesion temperature by 5°C each time, followed by repeat sensory testing. With progressive thermal neurolysis, these further lesion cycles can be facilitated by the administration of an analgesic (sufentanil or alfentanil) and often further propofol boluses will not be necessary. The process of sensory testing and lesion generation can be repeated until the desired level of hypalgesia is achieved.

■ Outcomes

There seems to be wide variation in the literature concerning the outcome of PRTG. Numerous reports describe what appear to be extended excellent outcomes after PRTG for TN.^{12–20} Many of these studies rely on the range and mean duration of follow-up to describe the patient population. Tew and Taha reported on a group of 1,200 patients followed up from 1 to 20 years (average follow-up of 9 years), and 93% had an excellent or good result, 4% were fair, and 1% rated poor.¹³ In contrast, Haridas et al²¹ have also reported their long-term results in 77 patients after PRTG for TN, and found that their 3-year success rate was 70.7%. In their series of 256 patients, this was compared with a 54.8% 3-year pain-free survival in 77 patients after glycerol injection, and 85.6% in 95 patients after MVD (microvascular decompression). Some authors have seen as much as a 53 to 80% recurrence rate when patients were followed long term.^{22,23}

The outcome from any pain surgery, including one for trigeminal neuralgia, can be measured by Kaplan–Meier statistics and reported as “pain-free survival.” This allows a prediction, over time, of the longevity of pain relief. In our experience, this method of analysis of the outcome of PRTG, and other procedures for trigeminal neuralgia, yields a somewhat less optimistic view of the durability of pain relief over time after PRTG.

“Pain-free survival” using Kaplan–Meier statistics was first described in 1981,²⁴ and since that time a number of investigators have followed suit for a wide range of pain procedures. The original report indicated that about 80% of patients were pain free immediately after PRTG, and that at 3 years postoperatively about 50% of patients experienced a full or partial recurrence (**Fig. 15.6**). In this series we saw a 15% incidence of nonbothersome paresthesias, a 4% incidence of anesthesia dolorosa, and a 3% rate of corneal anesthesia and keratopathy. This review involved contemporaneous follow-up of patients whose procedures had been performed up to 6 years previously, during the 1970s. What sets this series apart somewhat from the rest of the reported literature on this topic was the lower initial success rate (less than 80%), which may have skewed the pain-free survival period somewhat lower than subsequent case series reports have found.

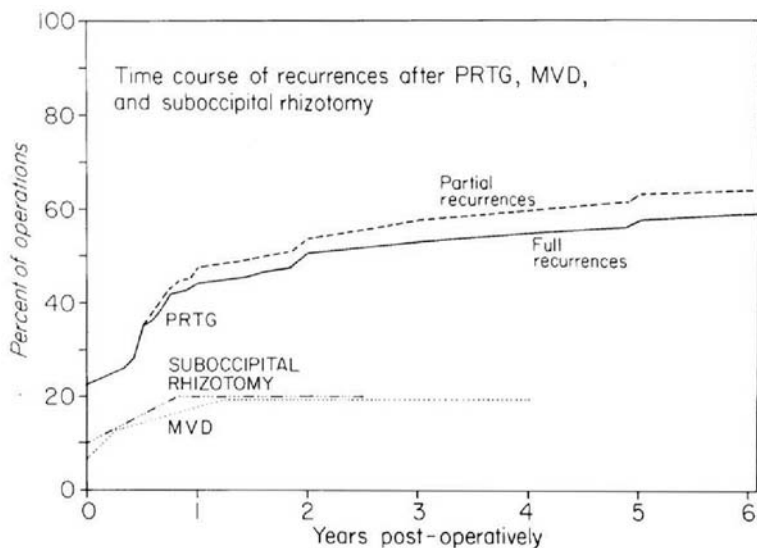


Fig. 15.6 First implementation of a Kaplan–Meier curve to describe “pain-free survival” after a surgical procedure. This graph shows the percentage of pain-free survival after PRTG (percutaneous radiofrequency trigeminal gangliolysis), MVD (microvascular decompression), and trigeminal rhizotomy, over time. (Reprinted from Burchiel et al.²⁴)

Taha et al¹⁸ employed Kaplan–Meier statistics in a series of 154 consecutive patients with trigeminal neuralgia treated by PRTG. Of the patients, 99% were pain free after their initial PRTG. They defined the sensory outcomes as analgesia (loss of pain perception without loss of touch perception), dense hypalgesia (loss of $\geq 75\%$ of pain perception, without loss of touch perception), and mild hypalgesia (loss of $< 75\%$ of pain perception, without loss of touch perception). Dysesthesia occurred in 23% of patients: 7% with mild initial hypalgesia, 15% with dense hypalgesia, and 36% with analgesia. Keratitis developed in 3 patients, and 14.3% had trigeminal motor weakness. The 14-year recurrence rate was 25% in the whole group: 60% in patients with mild hypalgesia, 25% in those with dense hypalgesia, and 20% in those with analgesia. The timing of the pain recurrence clearly varied according to the degree of sensory loss. All pain recurrences in patients with mild hypalgesia occurred within 4 years of surgery. The median pain-free survival rate was 32 months for patients with mild hypalgesia, and more than 15 years for patients with either analgesia or dense hypalgesia.

In comparing these results¹⁸ with our series,²⁴ it seems likely that our goal for the production of facial sensory loss from the PRTG procedure was closer to what these authors termed “mild hypalgesia” rather than to either “dense hypalgesia” or “analgesia.”

Lopez and colleagues have published a systematic review of ablative neurosurgical techniques for the treatment of trigeminal neuralgia, including PRTG.²⁵

Incorporating detailed and explicit inclusion criteria, this article is probably the best review of ablative procedures for TN to date. By their analysis, the success rate for PRTG was between 53 and 69% at 3 years, between 56 and 60% at 4 years, and between 51 and 56% at 5 years. In this survey, masticatory weakness occurred in 11.9%, cranial nerve deficits in 0.9%, meningitis in 0.2%, troublesome dysesthesias in 3.7%, anesthesia dolorosa in 1.6%, corneal numbness in 9.6%, and keratitis in 1.3%.

More recently, Udupi and colleagues²⁶ have reported on 39 patients after PRTG for TN, using the Kaplan–Meier method. They found that the pain-free survival probability at 54 months was 45.3%.

There seems to be no question that the amount of sensory loss produced by PRTG is directly related to the longevity of the pain relief, but also directly related to the incidence of facial dysesthesias or frank anesthesia dolorosa.¹⁸ The density of hypesthesia produced by the procedure may account for some of the variability in outcomes in both duration of pain-free survival and painful dysesthesias, but some of the variance is also likely due to the reporting method.

Without quantitative sensory testing, it is difficult to gauge exactly how much sensory loss has been produced by a PRTG procedure, or how that is communicated in the literature on the topic. It seems clear that an increased rate of dysesthesia is the price to be paid for an increased duration of relief from trigeminal neuralgia. It appears that there is considerable variability in what constitutes the goal of sensory loss among centers. Based on the most recent data using appropriate statistical reporting, the pain-free survival after PRTG with the goal of facial hypalgesia, but not analgesia, seems to be approximately 50% at 5 years postoperatively.

■ Conclusion

PRTG is an important option for pain control in trigeminal neuralgia. Although the pain-free outcome from surgery is less durable than for MVD, serious complications occur in less than 10% of procedures. It can be performed on outpatients, and it can be repeated, if necessary. The training and experience necessary to perform these procedures effectively and safely should be preserved in our residency training programs.

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Percutaneous Retrogasserian Glycerol Rhizolysis

Neal Luther, Douglas Kondziolka, and L. Dade Lunsford

The goals of surgical intervention for trigeminal neuralgia are rapid and long-lasting pain relief together with preservation of trigeminal nerve function. Percutaneous retrogasserian glycerol rhizotomy (PRGR) is a safe, minimally invasive approach that has been well established in the treatment of trigeminal neuralgia. In this chapter, we review our experience using glycerol rhizotomy in 1,174 patients to evaluate procedural technique, results, and complications. We further discuss the role of PRGR in the management of trigeminal neuralgia, along with other therapeutic options including medication, microvascular decompression, and Gamma Knife (Elekta, Stockholm, Sweden) radiosurgery for idiopathic or multiple sclerosis-related trigeminal neuralgia.

Trigeminal neuralgia is a challenging pain syndrome with numerous medical and surgical therapeutic options. The pain is pathophysiologically related to direct vascular compression of the trigeminal nerve in the majority of cases, with multiple sclerosis the next most common underlying etiology. Surgery is usually considered for patients who do not tolerate or respond adequately to medical treatment. Surgical interventions include microvascular decompression, which may directly remove the vascular etiology of pain if present, and ablative procedures that address nerve irritability. The ablative techniques include nerve balloon microcompression, thermal-induced axonal degeneration by radiofrequency rhizotomy, radiation-induced degeneration produced by stereotactic radiosurgery, and chemical ablation via percutaneous retrogasserian glycerol rhizotomy (PRGR). Injection of chemical agents to peripheral nerve targets (for example, alcohol injections to the supraorbital and infraorbital nerves) may also be performed. Radiosurgical ablation to the anatomical location of vascular compression may also work to “treat the cause.” However, it is believed that the effect is more likely a result of selective axonal degeneration of the nerve.

Approaching the trigeminal nerve less invasively through the face has a long history. Härtel is given credit for the accepted technique of spinal needle placement into the trigeminal cistern.¹ When absolute alcohol was injected into this location, multiple severe cranial neuropathies could be seen. Jefferson advocated the use of phenol mixed with glycerine rather than absolute alcohol.² Lars Leksell had long been interested in the use of focused radiation for the management of trigeminal neuralgia. His initial work in the early 1950s coupled an orthovoltage X-ray tube to a stereotactic frame to irradiate the trigeminal ganglion. The first-generation Gamma Knife was built in 1967. Leksell and Håkanson injected tantalum dust mixed with glycerol into the trigeminal cistern as a marker to localize the nerve for radiosurgery.^{3,4} When the targeting solution was injected prior to performing radiosurgery, patients noted pain relief. It was in this manner that PRGR was discovered as a therapeutic option.

In the care of patients with trigeminal neuralgia, we seek the goals of rapid onset and long-lasting pain relief together with preservation of trigeminal nerve function. PRGR offers distinct advantages over the aforementioned other percutaneous procedures. These include the lack of need for intraoperative confirmatory sensory testing (patient cooperation is not necessary) or for a radiofrequency generator. Precise anatomical localization of the target is established using intraoperative cisternography rather than having the patient describe radiofrequency-induced sensory changes. The patient does not need to participate during the procedure and, as a result, can be more deeply anesthetized. Glycerol is associated with a lower risk of facial sensory loss compared with either radiofrequency rhizotomy or balloon microcompression. This feature significantly reduces the risk of deafferentation pain. The choice of this procedure is in part related to the factors of patient age, medical condition, symptom severity, and personal preference. Finally, glycerol rhizotomy remains our preferred primary surgery for patients with multiple sclerosis–related trigeminal neuralgia.^{5,6}

■ Patient Selection

All surgical interventions for trigeminal neuralgia patients are decidedly more effective in patients without features of atypical facial pain. The correct diagnosis of trigeminal neuralgia is validated by a careful history that elucidates the quality, character, and distribution of pain. Patients with atypical trigeminal neuralgia may complain of a lingering, dull, or aching pain without triggers. In these patients, rhizotomy may be used to manage any severe lancinating component, but will likely have little effect on more constant pain. We rarely perform rhizotomy in this setting unless it is for patients who have undergone a successful procedure for prior typical trigeminal neuralgia symptoms.

We recommend initial medical treatment with appropriate doses of carbamazepine, oxcarbazepine, gabapentin, phenytoin, baclofen, or perhaps lamotrigine. Selected additional medications also may be used. High-resolution brain imaging should be performed to exclude a skull base lesion, a vascular anomaly, or a basal tumor that might change the regional anatomy or be the cause of trigeminal neuralgia. PRGR can be offered as first-line therapy in patients with idiopathic trigeminal neuralgia. However, we typically recommend Gamma Knife radiosurgery first unless the patient cannot eat or drink and requires more rapid pain relief. For such patients the possible latency interval associated with radiosurgery is less acceptable.⁷ As a minimally invasive strategy, PRGR is used as a second-line approach in patients who have not responded adequately to radiosurgery. Finally, PRGR is an excellent first choice for patients with trigeminal neuralgia in the setting of multiple sclerosis,^{5,6,8} which can occur at an earlier age than in patients without idiopathic neuralgia. It is an appropriate second-line therapy in those who have failed other surgical options such as microvascular decompression.

The technique of glycerol rhizotomy can vary from institution to institution. Some surgeons do not perform a contrast cisternogram, and others do not directly visualize the injection of glycerol into the trigeminal cistern with a metallic marker such as tantalum. Thus, if a prior attempt at PRGR failed elsewhere due to technical difficulties, we may repeat the procedure in an attempt to confirm that

glycerol was in fact injected in the appropriate location. Injection of contrast to identify the trigeminal cistern is the most conclusive way to know that the appropriate target has been reached.

Preoperative testing to rule out a bleeding diathesis, electrocardiogram, and chest X-ray are obtained as indicated. All antiplatelet agents (such as aspirin and ticlopidine) must be discontinued at least 1 week before PRGR. If patients must remain on warfarin or other anticoagulants, then radiosurgery is a better option.

■ Anesthetic Technique

At our institution, PRGR is performed with the patient under continuously monitored deep intravenous sedation in the operating room setting. The anesthesiologist monitors vital signs, provides adequate sedation, and is asked to quickly respond to any cardiovascular changes that might occur during the procedure. Patients generally receive propofol or another rapidly acting agent together with a narcotic. Some patients, especially younger men, may benefit from preadministration of 0.4 mg of atropine sulfate, which serves to blunt the occasional vasovagal response that may be seen during the procedure. Most of the discomfort during the procedure occurs when the 20-gauge spinal needle is passed through the foramen ovale, and it is thus important that patients are adequately sedated during this time. A deep injection of local anesthetic is as important as making sure that the skin surface is anesthetized.

Both the surgeon and anesthesiologist should be aware that up to 20% of patients can have a vasovagal response to transovale needle penetration or to the glycerol injection. Administration of an intravenous anticholinergic agent is important at the first sign of bradycardia. Other patients may have a hypertensive response to needle placement, usually due to pain or anxiety. Such a response can be lessened by the administration of hydralazine or beta-blockers. The systolic blood pressure should be kept below 160 mm Hg: higher blood pressures can be associated with facial hematomas from needle placement. Because the procedure is started with the patient supine but completed with the patient in the semisitting position during glycerol injection, a balance of pain control, blood pressure management, and respiratory care must be maintained by the surgeon and anesthesiologist.

■ Surgical Technique

The patient is placed supine on an operating room table that allows control of head, leg, and body position. The patient's head is suspended in a Mayfield cerebellar headrest (Mayfield Clinic, Cincinnati, OH, USA) so that the arm of the rest does not interfere with fluoroscopic imaging in the lateral or anterior-posterior (AP) direction. Initially, a C-arm fluoroscopic image intensifier is positioned in the AP projection. The head is aligned such that the petrous ridge is at the same level as the inferior orbital rim. The foramen ovale can often be visualized just inferior and lateral to the junction of the inferior and medial orbital rims. The face is sterilized with 70% ethanol solution, and towels are placed around the neck and upper chest as the area is draped.

The entry point, located 2.5 cm lateral to the corner of the mouth on the side of pain, is marked. From this point, straight-line trajectories toward the medial ipsilateral pupil and to a point 2.5 cm anterior to the external auditory canal are marked. Intradermal injection of lidocaine 1% with a 25-gauge needle is performed, and then a 21- or 23-gauge needle is used to inject lidocaine into the deep structures of the cheek. A gloved finger is placed inside the patient's oral cavity to prevent penetration of the mucosa either by the anesthetic needle or by the 20-gauge spinal needle used for the rhizotomy.⁹ The 20-gauge spinal needle is then inserted along the marked trajectory under fluoroscopic guidance toward the skull base. In the AP projection, the needle is guided toward the medial aspect of the inferior orbital rim (**Fig. 16.1**). Proper lateral positioning of the C-arm can be ensured by confirming that the auditory canals overlap (**Fig. 16.2**). Using the lateral projection, the desired trajectory is one directed toward the angle created by the clivus and petrous ridge (**Fig. 16.3a**).

Penetration of the foramen ovale can be uncomfortable for the patient, and thus administration of a short-acting barbiturate or propofol is administered immediately prior to puncture. Penetration of the needle through the foramen ovale can be felt by the surgeon. The stylet of the needle should be removed to check for flow of cerebrospinal fluid (CSF). If no CSF is encountered, then the needle with the stylet replaced is advanced at 1-mm increments under fluoroscopic guidance until the trigeminal cistern is entered. CSF flow should then be confirmed. If the needle has passed the clival line without flow of CSF, it may require adjustment; the most common problem is that the needle is either too lateral in the cistern or too medial. If too lateral within the foramen ovale, the tip of the needle may be in the subdural or subtemporal space. Although the finding of CSF flow is desirable, its absence does not always preclude identification of the trigeminal cistern (particularly in repeat cases). If CSF flow is identified or the needle is believed to be within the trigeminal cistern, the patient is placed in the sitting position for



Fig. 16.1 Intraoperative anteroposterior projection demonstrates spinal needle directed toward the inferomedial aspect of the orbit.



Fig. 16.2 Proper lateral position of the C-arm is confirmed by overlap of the auditory canals (arrow).

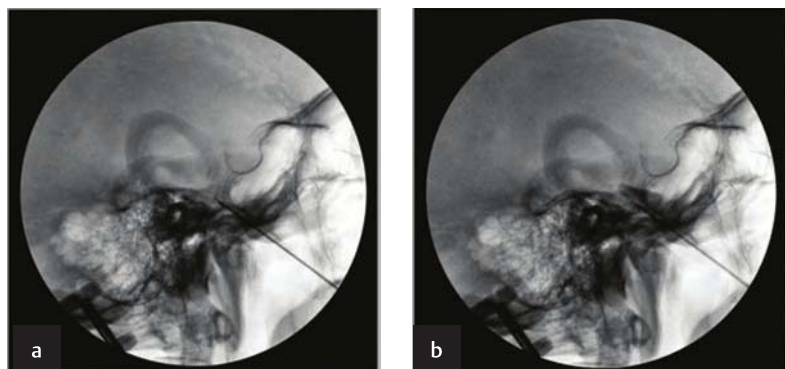


Fig. 16.3 (a) Intraoperative lateral projection shows spinal needle inserted into foramen ovale at the angle of the clivus and petrous bone. (b) Accurate placement is confirmed following injection of contrast, which fills the cistern in a characteristic pattern.

a contrast cisternogram. Cisternography is required to assess the volume of the trigeminal cistern and to select the proper amount of glycerol. With the needle in the cistern, the head of the bed is elevated to put the patient in the semisitting position with the neck slightly flexed. We use a tuberculin syringe to inject sterile iohexal in 0.05-mL increments and fluoroscopic guidance until the contrast is seen to overflow out of the cistern. The average volume of the trigeminal cistern is 0.25 mL, and the volume rarely exceeds 0.4 mL (**Fig. 16.3b**). The characteristic appearance of the cistern is of a bowling pin on its side. The contrast is then allowed to evacuate from the cistern by spontaneous drainage (which may require that the patient be placed again in the recumbent position). If full evacuation of the contrast is desired, particularly for patients with lower division pain, the patient can be returned to the supine position.⁹ It should be noted that some surgeons do not inject contrast,⁶ relying on fluoroscopic findings and CSF return. We continue to advocate contrast injection because it is the only way to directly confirm that the trigeminal cistern has been entered.

The glycerol injection is performed in the same manner as injection of contrast medium under fluoroscopic guidance. Again, the patient is placed in a semisitting position. The mixture used for ablation is 99.9% anhydrous glycerol with radiopaque tantalum powder. The final volume of glycerol injected is dependent upon the cisternal volume measured and the nerve distributions affected. There are different techniques for glycerol injection that vary from filling the entire cistern for patients with multidivision pain to leaving approximately one third of the contrast material still in the cistern and “floating” the glycerol on top of the contrast in patients with isolated V1 pain. Because of its lighter density, the glycerol mixture floats above the residual contrast medium, exerting its effect mainly on upper-division fibers. During the injection, some patients experience ipsilateral periorbital discomfort and facial flushing. Bradycardia is often seen and is further confirmation that the target has been localized. After the glycerol is injected, the needle is removed, and a small sterile adhesive strip is placed on the skin entry point. The patient remains in the semisitting position for 2 hours to prevent the

escape of glycerol into the posterior fossa. Most patients are kept in the hospital overnight because of the slight possibility they may experience headache from aseptic meningitis, and are discharged home the next day.

Many patients have chronic herpes simplex perioralis virus dormant in the gasserian ganglion. With percutaneous therapies, the subsequent development of cold sores is common. Patients with cold sore history are placed prophylactically on acyclovir and given acyclovir ointment in the perioperative interval. Early aseptic meningitis (1 or 2 days) is an extremely rare event, occurring in approximately 2 out of 1,000 cases. Although spinal fluid analysis should be repeated on an urgent basis to confirm the absence of bacterial meningitis, patients can be placed on corticosteroids if the CSF Gram stain is negative. The pleocytosis may be profound when it occurs, and can be very hard to differentiate from true bacterial meningitis. The risk of bacterial meningitis is minimized by making sure the spinal needle does not penetrate the oral mucosa.

■ Outcomes

There have been numerous studies that confirm the value of PRGR in the management of trigeminal neuralgia.^{4,6,8–16} Variations in surgical techniques do somewhat compromise efforts to compare the efficacy of PRGR with that of other treatment modalities. As previously mentioned, some centers do not perform cisternography, whereas others inject larger glycerol volumes. Most centers report that initial pain relief is seen in the majority of patients, with the rate of improvement in the range of 90%. We usually tell patients that half of those who respond will do so within the first day of the procedure, and in half it may take up to 2 or 3 weeks for the full effect. Some complexities exist as to the definition of recurrence rates depending on the time frame of evaluation, the need for concomitant medication, and the degree of pain control.

At the University of Pittsburgh, glycerol rhizotomy had been performed in 1,174 patients up to 2004. Immediate or early complete pain relief occurred in 90% of patients. An initial report that evaluated 112 patients demonstrated 90% pain relief at 2 years. Of these patients, 60% had complete relief after glycerol rhizotomy alone and 23% required additional (reduced) drug therapy. A subsequent analysis of 376 patients with follow-up to 7 years found a long-term pain control rate of 85%.¹⁰ Sixty percent had complete relief after glycerol rhizotomy alone, although in some patients repeat procedures were necessary. A longer term assessment up to 11 years found long-lasting relief of pain in 77% of patients, with 55% off all medications and 22% requiring some drug usage.¹⁰

It should be emphasized that trigeminal neuralgia is not a static disease but is characterized by remissions and recurrences, some mild and some severe. This challenge is known and expected at all experienced centers. Pollock recently reported on 98 patients and found that 73% were without pain at some point after surgery, with the chances of remaining pain free off medications found to be 61% and 50% at 1 and 3 years, respectively.¹³ Mild paresthesias or numbness was noted in 53% of patients, and 12% developed herpes simplex perioralis with full recovery.

Treatment of trigeminal neuralgia in the setting of multiple sclerosis poses a greater challenge. In these cases, microvascular decompression is not a viable option. A newly published report from Johns Hopkins of 822 patients with

trigeminal neuralgia directly compared the efficacy of PRGR with that of radiofrequency ablation in those with and without multiple sclerosis. Multiple sclerosis was present in 67 patients. This study did not find any significant difference in pain control outcomes (both rate of control and time to failure) between the two techniques in either setting. In patients with multiple sclerosis, pain control was achieved by PRGR in 68% of cases. Median time to failure was 25 months.¹⁶ Patients who underwent radiofrequency ablation were more likely to experience postoperative numbness than those who had PRGR.

Other than the perioperative blood pressure or cardiac changes noted above, other complications are relatively rare. Days after their first procedure, approximately 10 to 20% of patients will have detectable but usually mild reduction in light touch or pin prick perception. This still compares favorably with a roughly 30 to 40% likelihood of hypesthesias associated with radiofrequency ablation.¹⁶ With repeated glycerol procedures, the incremental sensory dysfunction assessment goes up, so that after two or three procedures, 50 to 70% of patients will have detectable sensory changes of a mild to moderate degree. Deafferentation

Editor's Comments

As the authors acknowledge, there are several options for the surgical treatment of trigeminal neuralgia. Percutaneous retrogasserian glycerol rhizolysis (PRGR) has some advantages and some disadvantages.

The procedure is fairly straightforward, although proper analgesia requires experience and coordination on the part of the surgeon and anesthesiologist. Bradycardia can occur during the penetration of the foramen ovale. During the injection of glycerol, the patient can experience severe burning pain in the ipsilateral trigeminal distribution. Profound bradycardia—even asystole—can also occur at this point. As the authors point out, prophylactic treatment with either atropine or glycopyrolate can prevent a crisis of bradycardia and hypotension.

The authors have a great deal of experience with PRGR, but for the uninitiated, the procedure can prove fairly daunting. Placement of the spinal needle with the patient in the supine position, under fluoroscopic control, using the Härtel technique can become routine with practice. However, at the point of cisternography and the glycerol injection, a fairly sedated patient is now placed in the sitting position with the head flexed forward, to allow first the contrast, and then the glycerol, to fill the cistern, and not pass immediately into the posterior fossa. If this sounds like a challenge, it can be!

I do agree that cisternography is important to verify the position of the needle tip in the cistern. Spinal fluid can be obtained from the subtemporal subarachnoid space, but this becomes apparent when the injected radiocontrast layers out in a thin stream along the floor of the middle fossa. Glycerol will work only if injected into the cistern and left in place for several hours while the patient remains sitting.

Finally, I am not quite as optimistic as the authors about the long-term relief of trigeminal neuralgia after PRGR. The literature generally supports a “half-life” of between 2 and 3 years. For multiple sclerosis, the results are even less durable. In our series, pain relief in these patients was for about 1 year.¹

The advantages of this procedure are that it does not require specialized equipment, pain relief is usually immediate or has a short latency, and the incidence of deafferentation pain is very low. Disadvantages include the need for technical expertise on the part of the surgeon and anesthesiologist, the potential for bradycardia and hypotension during the procedure, and the limited duration of pain relief.

As a tool, PRGR will remain solidly in the toolbox of neurosurgeons who treat trigeminal neuralgia.

pain in our experience has been extremely unusual and most likely occurs in the context of a complication noted below. The risk of developing delayed corneal dysfunction is extremely low, especially with an initial procedure.

In our experience with more than 1,000 patients with PRGR, we have had 1 perioperative death. This patient sustained a myocardial infarction about 1 hour after the procedure. This 0.1% surgical mortality risk should also take into account the consideration that this procedure was often undertaken in a higher risk population. For this population and others with this chronically debilitating pain syndrome, PRGR represents a safe and effective, minimally invasive treatment option.

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Percutaneous Balloon Compression for Trigeminal Neuralgia

Jeffrey A. Brown, Nathaniel D. Stetson, and Cletus Cheyuo

Three decades ago Sean Mullan published the results of his first 50 patients' treatment with "percutaneous microcompression of the trigeminal ganglion."¹ Although the operation is no longer considered to be either a "micro" compression or trigeminal "ganglion" compression, it has become a worldwide mainstay for the treatment of trigeminal neuralgia.

The purpose of this chapter is to review the indications for treatment, the nature of the injury caused by the procedure, the results and complications of treatment across many centers, and our recommendations regarding the technique for performing the procedure.

■ Nature of the Injury and Indications

Balloon compression selectively injures the myelin of large myelinated axons. It selectively preserves from injury small ganglion cells and fine-caliber primary efferent fibers.² Because the unmyelinated fibers that mediate the blink reflex are preserved, there is less chance of injuring the corneal reflex with balloon compression than with radiofrequency rhizotomy.³

The operation is effective because the trigeminal nerve is compressed within the anatomical confines of the porus trigeminus. The body of the balloon lies on the trigeminal ganglion. The ganglion is not a contained anatomical site; it is an open box demarcated by the temporal bone ventrally, and the subarachnoid and subdural spaces dorsally. When properly inflated, the balloon elevates the arachnoid and dura off of the ganglion. This requires some pressure, but it is not sufficient to effectively injure myelin or ganglion cells.² Histopathological studies of the ganglion in New Zealand rabbits after balloon compression show preservation of the ganglion cells at pressures sufficient to cause injury to large and medium-size myelinated fibers.²

When the balloon is inflated within the porus trigeminus, the trigeminal nerve root is compressed against the ventral petrous bone and the lateral and superior firm edge of the tentorial dura that splits to allow passage of the trigeminal root to the prepontine cistern. This forms the tip of the pear shape (**Fig. 17.1**). If the balloon is inserted farther, the fluoroscopic image seen will be a dumbbell shape wherein the balloon tip lies in the prepontine cistern.

Indications

Balloon compression is uniquely indicated for the ablative treatment of trigeminal neuralgia with first-division pain. Other indications are consistent with those of

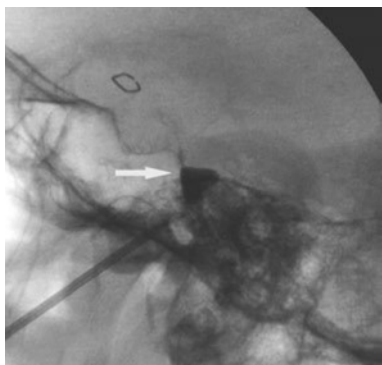


Fig. 17.1 Lateral radiograph of the skull. Arrow points to a properly inflated balloon. The tip forms the pear shape and is located within the entrance to Meckel cave, the porus trigeminus.

torial meningioma may cause trigeminal neuralgia pain by compressing a vessel against the trigeminal nerve. The presence of tumor in the pontine cistern usually will not interfere with completion of the procedure. A trigeminal schwannoma will likely cause dysesthesias and not paresthesias and may contraindicate balloon compression, although it will be technically possible to perform it. However, a tumor in Meckel cave will preclude successful compression. The balloon will not pass to the porous trigeminus.

Often older patients are on blood thinners such as warfarin, Lovenox, (Sanofi Pharmaceuticals, Bridgewater, NJ, USA) Plavix, (Sanofi Pharmaceuticals) or aspirin. Balloon compression is an intracranial procedure and has associated mortality. The medications must be stopped and their effect normalized before surgery. Often the decision to do this must be made in consultation with a cardiologist to learn the appropriateness of doing so and how soon after surgery a medication should be restarted.

Balloon compression causes brief but significant bradycardia and potential hypotension or hypertension.⁵ These changes can be rapidly limited, but patients should be able to tolerate brief variations. If a patient has a permanent pacemaker, its effectiveness should be confirmed preoperatively and it should be on during the surgery to provide protection from the bradycardia.

other percutaneous procedures. When constant dysesthetic pain predominates, balloon compression may not be indicated. The additional sensory loss caused by compression may aggravate the dysesthetic pain. Many clinical series have been published using balloon compression for the neuropathic facial pain associated with multiple sclerosis.⁴ Balloon compression may be done bilaterally, although not during the same anesthetic.

Preoperative Evaluation

Preoperative MR or CT imaging will show any associated tumor or arteriovenous malformation. These may not be a contraindication to balloon compression. An acoustic neuroma or ten-

torial meningioma may cause trigeminal neuralgia pain by compressing a vessel against the trigeminal nerve. The presence of tumor in the pontine cistern usually will not interfere with completion of the procedure. A trigeminal schwannoma will likely cause dysesthesias and not paresthesias and may contraindicate balloon compression, although it will be technically possible to perform it. However, a tumor in Meckel cave will preclude successful compression. The balloon will not pass to the porous trigeminus.

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■ Technique

Many articles on balloon compression speak about using the technique first described by Mullan and Lichtor. This is no more appropriate than attempting to do a microvascular decompression using the technique first described by Walter Dandy. Many advances in technique have been made in the ensuing decades. The original article may be of historical interest, but need not now be a technical reference.

The operation is done using general anesthesia, most commonly propofol and an inhalation agent. We do not give anticholinergic medications. The brief brady-

cardia that occurs during insertion of the cannula at the foramen ovale and during compression of the trigeminal nerve can be blocked with an external pacemaker once it occurs. It is helpful to obtain the feedback of the trigeminal depressor response that occurs during trigeminal nerve injury.⁵ This confirms that the nerve has been adequately compressed and does not endanger the patient. The pacemaker should be set to capture at 40 beats/minute and should be tested after induction with anesthesia. A patient with a functioning implanted pacemaker will not need the external pacer. Patients taking a beta-blocker as an antihypertensive may not develop bradycardia, but they should still be fitted with the external device. It is not necessary to insert an arterial catheter to monitor blood pressure changes. The blood pressure can be monitored with an inflatable cuff at 1-minute intervals during the period surrounding the compression. If bradycardia persists, then intravenous (IV) anticholinergic reversal can be given.

Preoperative antibiotic coverage is given intravenously, usually cephazolin. Patients with multiple sclerosis are given intravenous steroids. We have not seen a benefit from treating potential postoperative cold sores with acyclovir. We do not recommend muscle relaxants. This allows a feedback twitch to occur in the jaw muscles when the foramen is engaged by the introducing cannula.

This is usually an outpatient procedure. The patient is positioned on his/her back with a supportive roll behind the neck, which is in the neutral position. The anesthesiologist is positioned on the side opposite the planned surgery. The anesthesiology equipment is positioned at chest level, leaving room for the fluoroscopy unit at the head of the bed and the digital monitor at waist level.

The operation can be done in the operating room or imaging suite using biplane fluoroscopy. Before starting, obtain confirming images in the lateral, modified submental, and anterior-posterior positions so that the images can rapidly be obtained when the surgery starts. The equipment required is manufactured by Cook Medical (Bloomington, IN, USA) and called the Brown Percutaneous Trigeminal Ganglion Microcompression Set. It consists of a 14-gauge cannula, a blunt obturator, a sharp obturator, straight and angled guiding stylets, a no. 11 blade, a short angled ruler, a no. 4 F catheter, and a latex balloon (**Fig. 17.2**).

We use an insufflation syringe and a digital monitor to measure intraluminal pressure. Many physicians perform the operation "according to the technique of Mullan and Lichtor." This is volume- and image-controlled balloon inflation, not pressure-controlled. The volume of dye injected into the balloon and catheter is usually 0.75 to 1.0 mL. Some physicians vary the volume to adjust the fluoroscopic image of the inflated balloon to their concept of the "ideal" appearance of the "pear shape." We find it more reasonable to control the intraluminal pressure while monitoring the inflated balloon appearance. During the period of inflation, adjustments must be made to the inflation volume to maintain the pressure. One may, however, develop a sense of the intraluminal pressure from the pressure needed to inflate the balloon with the tuberculin syringe. We find it more repeatedly accurate to measure the pressure digitally in atmospheres. Our goal is to achieve 1.5 to 1.6 atmospheres of pressure for 1 to 1½ minutes.

Position the fluoroscopy unit so that the target screen is on the side opposite to the surgeon. The surgeon stands on the side of compression. First, obtain a pure lateral view by aligning the floor of the frontal fossa on the image. Second, obtain a modified submental view (**Fig. 17.3**). Set the screen of the fluoroscopy unit almost



Fig. 17.2 Disposable equipment used in performing percutaneous balloon compression. There is a no. 4 balloon catheter, a tuberculin syringe, a straight and an angled guiding stylet, a sharp and a blunt obturator, and a 14-gauge cannula.

on the chest of the patient and rotate the neck about 15 degrees away from the side of surgery. Find the foramen ovale just above the petrous ridge, medial to the mandibular head, and lateral to the maxillary sinus. Third, rotate the fluoroscopy unit to obtain a modified anterior-posterior view (**Fig. 17.4**). This view centers the petrous ridge in the orbit as viewed radiographically. By maintaining the 15-degree rotation of the neck it is possible to see the dip in the petrous ridge that represents the porus trigeminus or the entrance to Meckel cave.

Return to the lateral view, prepare the face with sterile solution, and mark a point 2.5 cm lateral to the angle of the lip for placement of the cannula. Surround this entrance point with sterile plastic drapes and put a drape across the body. The 14-gauge cannula with a sharp obturator is inserted at a point in the cheek that places it along the line extending from the floor of the petrous ridge. That may require adjusting the point of insertion above or below the standard Haertel point lateral to the angle of the lip. If the target is the first division, then the cannula is inserted just below and lateral to Haertel point. This



Fig. 17.3 Modified submental fluoroscopic image. The stylet just penetrates the foramen ovale, which is seen superior to the petrous ridge, medial to the head of the mandible, and lateral to the maxillary sinus.

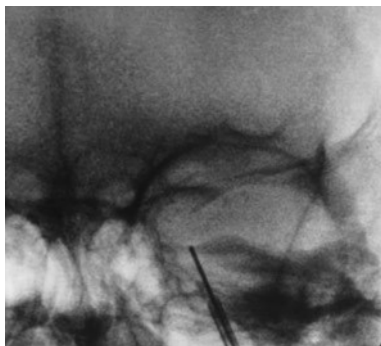


Fig. 17.4 Modified anterior-posterior radiograph of the skull with the petrous bone aligned in the center of the orbit as viewed radiographically. The balloon tip is positioned in the center of the dip for optimal compression of second-division trigeminal pain.

allows direction of the balloon tip more superomedial so that the fibers of the first division are selectively compressed. This angle makes it more difficult to engage the foramen ovale with the cannula, however.

Make a stab incision with the no. 11 blade. Insert the cannula and inner sharp obturator through the cheek skin using the lateral fluoroscopic guidance. Once the cannula has penetrated the cheek skin, switch from the sharp to the blunt obturator. The cannula need not engage the foramen ovale at this point and should be positioned at the level of the skull base. By using the blunt obturator, the risk of arterial injury is eliminated.

Switch to the modified submental view to see the foramen ovale. Direct the cannula to the center of the foramen ovale, but only to engage it. A brief drop in heart rate will occur when the foramen is engaged and the mandibular branch stimulated. There may be a twitch from the masseter muscle also.

Remove the blunt obturator and insert a straight guiding stylet. Switch to the modified anterior-posterior view. For second-division pain direct the guiding stylet to the midpoint in the dip in the petrous bone representing the entrance to Meckel cave, then 2 mm beyond. For isolated third-division pain it may be useful to direct the stylet slightly more lateral. For first-division pain, the stylet is directed lateral to medial. On the lateral view the angle away from the petrous bone will be wider. Usually it is simplest to direct the stylet and balloon to the midpoint in the dip. Remove the stylet and place the balloon catheter in the same location. The kit is designed so that the second mark on the catheter is within a millimeter of the desired distance from the foramen ovale (17 mm). There is a slight give when the catheter enters the porus trigeminus.

The anteroposterior (AP) view is essential to obtain. It is possible to believe that the catheter is within the porus when it is actually more lateral and positioned beneath the temporal lobe rather than adjacent to the trigeminal nerve. In that situation, the balloon will not achieve the pear shape that is the most significant association with a successful operation. The “pear” is the portion of the balloon within the tunnel created by the split in the tentorium to allow the nerve to pass from the middle to the posterior fossa through the prepontine cistern. A more medial position of the catheter is possible also. In this situation the catheter passes through the inferior orbital fissure. If inflated, it may injure the optic nerve (**Fig. 17.5**). By combining the use of these three images, the likelihood of successful balloon compression is increased.

Switch to the lateral view. Confirm the balloon catheter position and that the tip of the balloon is posterior to the clival line. Remove the inner stylet. Attach a three-way stopcock. Fill the insufflation syringe with radiopaque dye. Make sure that there are no air bubbles in the insuffla-

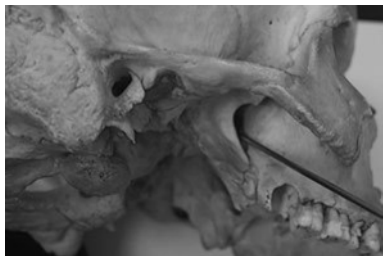


Fig. 17.5 Photograph of a skull with the 14-gauge cannula inserted into the inferior orbital fissure instead of the adjacent foramen ovale. If only a lateral radiograph is used during surgery, it may seem that the cannula is properly positioned at the foramen ovale when it is not.

tion syringe line. Attach the syringe to the balloon and remove air from the catheter with the tuberculin syringe. Attach the insufflation syringe to the digital monitor and allow the reading to stabilize.

Switch the blood pressure cuff to stat mode. Ask the anesthesiologist to control the rise in blood pressure expected during compression. While using intermittent lateral fluoroscopic imaging, slowly inflate the balloon to a pressure of 1.5 atm (atmosphere of pressure) and observe for the “pear” shape. Patients requiring repeat compression or who have severe pain may need 1½ minutes of compression at 1.6 atm rather than 1.5 atm compression for 1 minute. The latter will almost always lead to mild sensory loss and preserve corneal sensation.

If the pacemaker is triggered, it is usually for only a few beats. Prolonged bradycardia can be treated with intravenous anticholinergic injection. It is rare for it to be required, but the injection should be readied.

After compression, deflate the balloon completely and remove it along with the cannula to prevent the creation of a tear in the balloon. If the balloon is removed before the cannula, slightly bloody cerebrospinal fluid (CSF) will drip and then clear. CSF will not drip when the cannula is first positioned because it does not penetrate the subarachnoid space at the level of the foramen ovale.

Compress the incision against the maxilla for 5 minutes to prevent bleeding in the cheek and place a simple bandage. In the recovery room use an ice pack to reduce swelling.

■ Technical Problems

Rarely (e.g., when there is Paget disease), the foramen ovale will not accommodate the 14-gauge cannula or allow penetration by the balloon catheter. Repeated balloon compression can make it difficult to inflate the balloon. Scar tissue may form and thicken the dura, increasing the pressure required to open the balloon. If inserted too far, the balloon may either inflate in the prepontine cistern or form a dumbbell shape from partial inflation in the cistern and partial inflation in the porus trigeminus. Neither of these configurations is likely to lead to adequate nerve compression. Repeated inflations that fail to lead to a pear shape require reconsideration of the balloon position. If the balloon is placed over Meckel cave, but not within the porus, inflation will elevate the dura off of the ganglion; there will be no pear shape. This requires some pressure, but it is insufficient to achieve lasting pain relief. If the guiding stylet tracks lateral or medial to the porus trigeminus after repeated attempts, the cannula should be removed and repositioned. Sometimes there will be venous bleeding when the cannula is positioned at the foramen ovale. This is usually from a venous complex at the skull base. The bleeding can be abated if the cannula is held against the foramen ovale.

■ Morbidity

Jaw weakness almost always occurs and almost always resolves within 1 month (Table 17.1). The incidence of painful dysesthesia/numbness in our series is 6%.⁶ Corneal anesthesia is reported. Because compression selectively preserves the

small myelinated and unmyelinated fibers that mediate the blink reflex, the corneal reflex is preserved. Meningitis has occurred, but is rare. Several unusual complications have been reported. Cavernous sinus injury has occurred with injury to vision. Most likely this was because of placement of the cannula in the inferior optic fissure. The procedure should be done using the multiple views described above so that misplacement of the cannula does not occur. Bleeding is more likely if a kit that has a sharp pointed obturator is used. The Brown access kit has a blunt obturator for use after the skin of the cheek is penetrated. Balloon rupture is possible, but no complications have been reported because of it. Allergy to dye could be pretreated with steroids, if it is known to be present. The volume of dye leaked by a ruptured balloon is small, however. There has been a death when the inflated balloon ruptured an undiagnosed fistula present after a previous microvascular decompression (MVD) but not seen on preoperative magnetic resonance imaging (MRI). Although “simple” to perform, the compression is intracranial and there is still significant risk to be considered.

■ Results

Published series dating from 1983 indicate a range of initial success rates from 64 to 100% (**Table 17.1**).^{6–24} Recurrence rates vary from 0%, in an early series, to 59% (in a series that used a compression time of 60–90 seconds and had a lower initial success rate of 83%). The lower initial success rate also occurred in an earlier series in which only 48% of patients had sensory loss from the compression. Skirving's series from 2001²⁰ had more than 500 compressions that were evaluated. The initial success rate was 96%. The recurrence rate was 32% when compression was done for a range of 2 to 7 minutes. This series achieved 89% sensory loss. Montano focused on a series of patients with multiple sclerosis. He had only 81% initial success in 21 patients, achieved only a 10% incidence of sensory loss, and had a 57% recurrence rate in a mean of 15 months.¹¹ Masseter and pterygoid muscle weakness, when reported, varied from 4 to 100%. Asplund et al demonstrated a significant correlation of success with the presence of a pear shape during compression ($p < 0.001$). There was no association with the presence of hypesthesia.⁷ Depression of the corneal reflex, when reported, was usually 2 to 3%. Chen et al reported an incidence of 16% when they compressed for an average of 4 minutes; however, these patients were all undergoing repeat compression.¹⁰ Only two series used pressure monitoring.^{6,14} Corneal anesthesia did not occur in these series.

Balloon compression for trigeminal neuralgia is a standard percutaneous treatment that is simple to perform. It is best indicated for treatment of first-division or multidivision trigeminal neuralgia when a percutaneous procedure has been selected. It is also applicable to patients with multiple sclerosis and it may be repeated when recurrence occurs. Care should be taken to adhere to technical guidelines when performing balloon compression to limit the potential morbidity and maximize the potential of successful pain alleviation.

Table 17.1 Published results of treatment of trigeminal neuralgia by balloon compression

A.	Author (year)				
	Chen et al (2012) ¹⁰	Montano et al (2012) ¹¹	Baabor et al (2011) ¹²	Chroni et al (2011) ¹³	Stomal-Słowińska et al (2011) ¹⁴
No. of patients/no. of PBCs	32/32 ^a	21/21	206/206	15/15	59/92
<i>Demographics</i>					
Age range	26–79	30–75	20–80	65–80	29–87
Gender (men:women)	14:18	10:11	74:132	10:5	23:36
Pain distribution: no. of patients					
V1	0	8	5	0	–
V2	18.7	10	25	26.7	–
V3	25	3	25	53.3	–
Multiple	56.3	0	151	20	47
Bilateral	0	0	–	0	0
Classification of TN: no. of patients					
Typical	32	0	–	–	42
Atypical					
TN2	0	0	–	–	7
Trig. neuropathic	0	0	–	–	2
TN in MS	0	21	2	–	3
Postherpetic	0	0	–	–	3
Trig. pain + complex etiology	0	0	–	–	2
No. of patients who had prior operation(s)					
MVD	–	0	22	–	6
RFT	–	2	9	–	9
GR	–	3	14	–	2
AR	–	0	–	–	3
SRS	–	1	–	–	5
PBC	32	1	–	–	0
Others (periph. block, etc.)	–	2	–	–	0
<i>Surgical parameters</i>					
Balloon shapes: no. of patients					
Typical pear	–	0	–	–	–
Others (pearlike, dumb bell, elliptical, etc.)	–	21	–	–	–
Duration of compression (seconds)	240	120–720	78–180	120–300	60–90

(Continued)

A.	Author (year)				
	Chen et al (2012) ¹⁰	Montano et al (2012) ¹¹	Baabor et al (2011) ¹²	Chroni et al (2011) ¹³	Stomal- Słowińska et al (2011) ¹⁴
<i>Outcome measures</i>					
Initial success rate (%)	93.8	80.95	93	100	83
Recurrence rate (%)	43.3	57.14	15	20	59
Mean time to recurrence (months)	–	15	–	6	–
Follow-up range (months)	60.96–105	30.71–72.46	1–48	1–12	3–48
<i>Morbidity (%)</i>					
Defined postoperative period (months)	< 3	None specified	> 2	< 1	12–82
Corneal anesthesia/ decreased reflex	15.6	0	–	0	0
Masseter/pterygoid weakness	18.8	0	–	100	11.9
Trigeminal depressor response	0	0	–	0	0
Dysesthesia	0	0	–	0	1.7
Hypesthesia	0	0	–	0	0
Hypoesthesia	62.5	9.5	–	0	90
Diplopia					
CN VI palsy	0	0	–	0	3.4
CN IV palsy	0	0	–	0	0
CN unspecified	0	0	–	0	0
Hearing loss/CN VIII	0	0	–	0	0
Blindness/CN II	0	0	–	0	0
Anesthesia dolorosa	0	0	0	0	1.7
Meningitis	–	–	–	0	0
Vascular complication					
Hematoma	0	0	0	0	0
CC fistula	–	–	–	0	0
IC hemorrhage	40.6	0	–	–	–
Herpes simplex oralis	0	0	–	0	0
Failure due to technical difficulties	0	0	–	0	0
Other	–	–	–	0	0
<i>Mortality (%)</i>	0	0	0	0	0

B.	Author (year)				
	Campos et al (2011) ⁸	Chen et al (2011) ⁹	Kouzounias et al (2010) ⁴	Kouzounias et al (2010) ¹⁵	Asplund et al (2010) ⁷
No. of patients/no. of PBCs	39/39	130/130	47/66	61/66	69/87
<i>Demographics</i>					
Age range	62.3 ± 12.5	26–83	34–91	60–80	43–88
Gender (men:women)	18:21	63:67	26:35	43:57	1:2.2
Pain distribution: no. of patients					
V1	2	3.8	3	26	–
V2	10	20.8	11	74	–
V3	8	19.2	23	72	–
Multiple	19	54.7	62	–	–
Bilateral	0	1.5	8	–	–
Classification of TN:					
no. of patients					
Typical	39	130	35	–	49
Atypical					
TN2	0	0	0	–	0
Trig. neuropathic	0	0	4	–	0
TN in MS	0	0	17	17	20
Postherpetic	0	0	0	–	0
Trig. pain + complex etiology	0	0	0	–	0
No. of patients who had prior operation(s)					
MVD	–	32	13	13	6
RFT	–	7	0	0	1
GR	–	0	51	50	18
AR	–	0	0	0	0
SRS	–	9	7	7	0
PBC	–	0	19	20	23
Others (periph. block, etc.)	–	14	0	0	0
<i>Surgical parameters</i>					
Balloon shapes: no. of patients					
Typical pear	–	117	18	–	46
Others (pearlike, dumbbell, elliptical, etc.)	–	13	43	–	41
Duration of compression (seconds)	–	70–90	65–180	60–120	60–180

(Continued)

B.	Author (year)				
	Campos et al (2011) ⁸	Chen et al (2011) ⁹	Kouzounias et al (2010) ⁴	Kouzounias et al (2010) ¹⁵	Asplund et al (2010) ⁷
<i>Outcome measures</i>					
Initial success rate (%)	79.5	93.8	85	85	–
Recurrence rate (%)	20	37.7	60.5	50	–
Mean time to recurrence (months)	36	–	17.28	21	–
Follow-up range (months)	≤ 50	96–121.2	5.5–50.5	0.5–48	4–73
<i>Morbidity (%)</i>					
Defined postoperative period (months)	–	≤ 3	≤ 4	–	–
Corneal anesthesia/ decreased reflex	2.6	2.3	2	3	–
Masseter/pterygoid weakness	17.9	6.2	–	7	–
Trigeminal depressor response	0	0	–	0	–
Dysesthesia	0	0	4.3	5	–
Hypesthesia	0	0	–	62	–
Hypoesthesia	84.5	30.7	–	0	–
Diplopia					
CN VI palsy	0	1.5	–	–	–
CN IV palsy	2.5	0	–	–	–
CN unspecified	0	0	–	–	–
Hearing loss/CN VIII	0	0	2	5	–
Blindness/CN II	0	0	–	0	–
Anesthesia dolorosa	0	0	–	0	–
Meningitis	0	0	–	0	–
Vascular complication					
Hematoma	2.5	2.3	–	0	–
CC fistula	0	0	2	3	–
IC hemorrhage	–	–	–	–	–
Herpes simplex oralis	43.6	33.1	–	7	–
Failure due to technical difficulties	0	0	–	0	–
Other	0	0	2	0	–
<i>Mortality (%)</i>	0	0	0	0	0

C.	Author (year)					
	Park et al (2008) ¹⁶	Omeis et al (2008) ¹⁷	de Siqueira et al (2006) ¹⁸	Brown and Pilitsis (2005) ⁶	Lee and Chen (2003) ¹⁹	Skirving and Dan (2001) ²⁰
No. of patients/no. of PBCs	50/50	29/41	105/105	56/65	80/80	496/531
<i>Demographics</i>						
Age range	27–83	26–90	35–85	37–92	45–86	18–86
Gender (men: women)	22:28	14:15	45:60	23:33	33:47	279:217
Pain distribution: no. of patients						
V1	4	7	5	10	0	–
V2	32	25	31	25	0	–
V3	18	23	31	30	80	–
Multiple	46	–	38	0	0	146
Bilateral	0	0	1	0	0	0
Classification of TN: no. of patients						
Typical	50	29	–	–	–	496
Atypical						
TN2	0	0	–	–	–	0
Trig. neuropathic	0	0	–	–	–	0
TN in MS	0	0	–	6	–	11
Postherpetic	0	0	–	–	–	0
Trig. pain + complex etiology	0	0	–	–	–	0
No. of patients who had prior operation(s)						
MVD	5	2	–	4	–	7
RFT	4	15	–	–	–	11
GR	1	1	–	–	–	–
AR	0	0	–	–	–	–
SRS	1	1	–	–	–	–
PBC	0	10	–	–	–	–
Others (periph. block, etc.)	6	0	–	30	–	–
<i>Surgical parameters</i>						
Balloon shapes: no. of patients						
Typical pear	33	29	105	56	–	–
Others (pearlike, dumbbell, elliptical, etc.)	17	0	0	0	–	–
Duration of compression (sec)	60–20	30–90	30–60	60–90	60–80	120–420

(Continued)

C.	Author (year)					
	Park et al (2008) ¹⁶	Omeis et al (2008) ¹⁷	de Siqueira et al (2006) ¹⁸	Brown and Pilitsis (2005) ⁶	Lee and Chen (2003) ¹⁹	Skirving and Dan (2001) ²⁰
Outcome measures						
Initial success rate (%)	92	83	99	92	100	99.8
Recurrence rate (%)	16	45	16.2	16	2.5–5	31.9
Mean time to recurrence (months)	18	17	2.5	12.6	12	128.4
Follow-up range (months)	12–82	1–101	1–6	3–38	1–12	1–204
Morbidity (%)						
Defined postoperative period (months)	–	–	< 3	–	< 12	–
Corneal anesthesia/ decreased reflex	0	52	–	0	0	0
Masseter/ pterygoid weakness	4	6.9	50.5	24	100	3.4
Trigeminal depressor response	34	–	–	–	0	–
Dysesthesia	0	6.9	5.2	4	0	3.8
Hypesthesia	0	–	–	0	0	–
Hypoesthesia	–	–	–	83	90	89
Diplopia						
CN VI palsy	–	–	–	0	0	–
CN IV palsy	–	–	–	0	0	–
CN unspecified	–	–	–	0	0	–
Hearing loss/CN VIII	–	–	7.6	0	0	–
Blindness/CN II	–	–	–	0	0	–
Anesthesia dolorosa	–	3.4	–	0	0	0
Meningitis	–	–	1	0	0	0
Vascular complication						
Hematoma	–	–	32.3	0	0	0.01
CC fistula	–	–	–	0	0	–
IC hemorrhage	–	3.4	–	2	0	–
Herpes simplex oralis	26	–	44.6	–	0	–
Failure due to technical difficulties	8	–	–	0	0	–
Other	–	–	–	0	0	–
Mortality (%)	0	0	0		0	0

D.	Author (year)				
	Abdennebi et al (1995) ²¹	Lichter and Mullan (1990) ²²	Lobato et al (1990) ²³	Fraioli et al (1989)	Belber and Rak (1987) ²⁴
No. of patients/no. of PBCs	150/150	100/100	144/164	159/159	25/33
<i>Demographics</i>					
Age range	24–84	–	30–90	–	48–86
Gender (men:women)	73:77	–	58:86	–	15:10
Pain distribution: no. of patients					
V1	0	–	5	–	–
V2	19	–	33	–	–
V3	14	–	39	–	–
Multiple	117	–	67	–	–
Bilateral	2	7	4	–	–
Classification of TN: no. of patients					
Typical	–	–	–	143	21
Atypical					
TN2	–	–	–	0	0
Trig. neuropathic	–	–	–	0	0
TN in MS	–	–	3	3	4
Postherpetic	–	–	–	0	0
Trig. pain + complex etiology	–	–	–	13	0
No. of patients who had prior operation(s)					
MVD	–	–	3	–	–
RFT	–	1	43	3	–
GR	–	–	–	10	–
AR	–	20	–	–	–
SRS	–	–	–	–	–
PBC	–	–	–	–	–
Others (periph. block, etc.)	–	–	27	–	–
<i>Surgical parameters</i>					
Balloon shapes: no. of patients					
Typical pear	–	100	117	–	–
Others (pearlike, dumbbell, elliptical, etc.)	–	0	27	–	–
Duration of compression (seconds)	300	300–420	60–180	60–420	240–600

(Continued)

D.	Author (year)				
	Abdennebi et al (1995) ²¹	Lichtor and Mullan (1990) ²²	Lobato et al (1990) ²³	Fraioli et al (1989)	Belber and Rak (1987) ²⁴
<i>Outcome measures</i>					
Initial success rate (%)	96	100	100	89.9	100
Recurrence rate (%)	30.6	28	9.7	9.8	24
Mean time to recurrence (months)	–	–	10–35	–	–
Follow-up range (months)	1–48	12–120	6–54	42	6–84
<i>Morbidity (%)</i>					
Defined postoperative period (months)	–	–	–	–	–
Corneal anesthesia/decreased reflex	2.6	0	–	–	–
Masseter/pterygoid weakness	6.6	–	12	10	48
Trigeminal depressor response	–	–	–	–	–
Dysesthesia	10.6	–	–	–	12
Hypesthesia	–	17	4.1	–	–
Hypoesthesia	93.4	–	40	76.7	100
Diplopia					
CN VI palsy	1.5	–	–	–	–
CN IV palsy	–	1	1	–	–
CN unspecified	–	–	–	–	–
Hearing loss/CN VIII	6.6	–	–	–	–
Blindness/CN II	–	–	–	–	–
Anesthesia dolorosa	–	0	0	0.6	–
Meningitis	–	–	1.4	–	–
Vascular complication					
Hematoma	–	–	–	–	–
CC fistula	6.6	–	3.5	–	16
IC hemorrhage	–	–	–	–	–
Herpes simplex oralis	–	–	–	–	–
Failure due to technical difficulties	–	–	11	–	48
Other	–	–	6.2	0.6	–
Mortality (%)	0	0	0	0	0

E.	Author (year)			
	Brown et al (1989)	Meglio et al (1987)	Esposito et al (1985)	Mullan et al (1983)
No. of patients/no. of PBCs	22/24	47/47	50/50	50/50
<i>Demographics</i>				
Age range	36–83	–	48–82	16–88
Gender (men:women)	16:6	–	19:31	28:22
Pain distribution: no. of patients				
V1	–	–	–	–
V2	–	–	–	–
V3	–	–	–	–
Multiple	–	–	13	–
Bilateral	–	–	–	–
Classification of TN: no. of patients				
Typical	22	–	–	48
Atypical				
TN2	0	–	–	0
Trig. neuropathic	0	–	–	0
TN in MS	0	–	–	2
Postherpetic	0	–	–	0
Trig. pain + complex etiology	0	–	–	0
No. of patients who had prior operation(s)				
MVD	2	–	0	5
RFT	–	–	0	2
GR	–	–	0	0
AR	–	–	0	16
SRS	–	–	0	0
PBC	–	–	0	6
Others (periph. block, etc.)	11	–	0	1
<i>Surgical parameters</i>				
Balloon shapes: no. of patients				
Typical pear	22	–	–	50
Others (pearlike, dumb-bell, elliptical, etc.)	0	–	–	0
Duration of compression (seconds)	60–180	240–360	300	180–600

(Continued)

E.	Author (year)			
	Brown et al (1989)	Meglio et al (1987)	Esposito et al (1985)	Mullan et al (1983)
<i>Outcome measures</i>				
Initial success rate (%)	100	100	64	–
Recurrence rate (%)	14	54.5	0	12
Mean time to recur- rence (months)	9.5	–	6	16.5
Follow-up range (months)	3–53	36	6	6–54
<i>Morbidity (%)</i>				
Defined postoperative period (months)	≤ 3	–	–	1–4
Corneal anesthesia/ decreased reflex	0	–	0	–
Masseter/pterygoid weakness	14	4.25	–	–
Trigeminal depressor response	–	–	–	–
Dysesthesia	14	8.5	0	–
Hypesthesia	23	–	–	6
Hypoesthesia	23	–	48	8
Diplopia				
CN VI palsy	5	–	–	–
CN IV palsy	0	–	–	2
CN unspecified	0	–	–	–
Hearing loss/CN VIII	0	–	–	–
Blindness/CN II	0	–	–	–
Anesthesia dolorosa	0	–	–	–
Meningitis	5	–	–	–
Vascular complication				
Hematoma	0	–	–	–
CC fistula	0	–	–	–
IC hemorrhage	0	–	–	–
Herpes simplex oralis	–	–	–	8
Failure due to technical difficulties	0	–	–	4
Other	14	–	–	–
<i>Mortality (%)</i>	0	0	0	0



Video 1. Percutaneous Balloon Compression for Trigeminal Neuralgia

Editor's Comments

Balloon compression continues to be an option for patients with refractory trigeminal neuralgia (TN). When performed properly, as described by Dr. Brown and colleagues in this chapter, the procedure should be safe and painless for the patient. The outcome is determined by the technique used, in that if the balloon is inflated and the characteristic “pear shape” is not seen on lateral fluoroscopy, the balloon can compress other structures within the cavernous sinus (e.g., CN VI) and produce neurologic complications.

In the correct radiographic position, filling of the balloon to produce nerve compression must be carefully accomplished and monitored. In the early days of this procedure, the balloon was filled to higher pressures than are currently employed, and held at pressure for longer times. This resulted in good relief of TN, but also a higher rate and density of facial hypesthesia, which resulted in a higher incidence of uncomfortable dysesthesias and even frank anesthesia dolorosa. The trend has been for the balloon inflation times to diminish, which has reduced consequent numbness, but also decreased the median time to recurrence of TN after the procedure.

Review of the table in this chapter shows that the median time to recurrence after balloon compression generally runs 1 to 2 years. Longer intervals of pain relief are seen in centers using longer compression times, and are associated with higher incidences of facial hypesthesia. The results of balloon compression from most centers compare favorably with those of glycerol injection, but are generally inferior to results from radiosurgery or radiofrequency lesions in terms of time to pain recurrence.

I would agree that balloon compression is a “mainstay” therapy for medically intractable TN, if the correct technique is used, and the expectations of the duration of pain relief are explained to the patient.

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18

Intracranial Procedures for Nontrigeminal Neuralgias

Nicholas Barbaro and Jason D. Hill

This chapter aims to examine the causes and surgical treatments of one small but not insignificant outpost within the landscape of pain disorders: nontrigeminal pain. We begin with a brief discussion of the relevant anatomy, followed by an overview of some of the more common nontrigeminal pain disorders. We then conclude with a survey of ablative and nonablative intracranial surgical techniques.

It is important to remember that pain in the distribution of a given cranial nerve or nerves may be due to extrinsic factors such as tumors, compression by adjacent intracranial structures, inflammation, and mass effect secondary to infection, or intrinsic factors such as ischemia. A thorough workup, including dental examination and ear, nose, and throat (ENT) evaluation, as well as appropriate imaging, should be undertaken to rule out other causes of nontrigeminal pain.

Keep in mind that a patient should exhaust all medical options prior to undergoing surgery. Common medications used to treat chronic and/or neuropathic pain include, but are not limited to, gabapentin, pregabalin, carbamazepine, amitriptyline, narcotic medications, as well as agents used in headache management, such as verapamil and the triptans.

■ Anatomy

The pain system is a complex biochemical and cellular environment, where noxious stimuli are first apprehended and reacted to via reflex pathways in the lower nervous system, are transmitted centrally where processing and reflex refinement take place, and are only then consciously felt by the individual. The conscious recognition of noxious stimulation we might term *pain*, whereas the continued psychic distress elicited by these pain signals might be termed *suffering*. At times, as in the case of chronic pain, the pain pathways may continue to fire in the absence of the original stimulus, resulting in continued suffering following the resection or removal of a clear anatomic lesion.

Pain signals travel through the dorsal horn/Lissauer tract and ascend to the thalamus via the anterolateral and lateral spinothalamic tracts.¹ The lateral spinothalamic tracts originate from lamina I and primarily transmit C fiber signals, whereas the anterolateral tracts have their origin in lamina IV and V and transmit A δ and A β signals.¹ The lateral spinothalamic tract may be further subdivided into paleospinothalamic and neospinothalamic divisions,² with the paleospinothalamic tract involved in the emotional dimension of pain.² These synapse in the lateral thalamus in the case of the neospinothalamic and anterolateral tracts (VPL/VPI), and the intralaminar nuclei and medial thalamus (mediodorsal nucleus/medial nucleus of the posterior group) in the case of the paleospinothalamic

tract. Although there is evidence that these neurons may also send terminations to the VPL/VPM/VPI, others hypothesize projections to a distinct nucleus termed VMpo.¹ VPM and VPL signals then travel to SI, whereas VPI signals transmit to SII. Mediodorsal signals that have their origin in the lateral spinothalamic tract project to the anterior cingulate; the medial nucleus of the posterior group transmits to the insula. Procedures such as medial thalamotomy and cingulotomy have sought to arrest chronic pain at these levels of processing.

Pain disorders often involve discrete cranial nerves, resulting in neuralgias of the trigeminal or glossopharyngeal nerves, as well as the nervous intermedius/geniculate ganglia. The nervus intermedius is a component of cranial nerve (CN) VII and is formed when fibers from the superior salivatory nucleus join fibers from the nucleus of the solitary tract.² Fibers from the facial nucleus then join nervus intermedius as they emerge from the pons. Nervus intermedius projects to the geniculate ganglion and is situated between cranial nerve VII motor fibers and cranial nerve VIII.² Of clinical relevance, the nervus intermedius contains parasympathetic as well as sensory fibers from the external auditory meatus, and is a target in the treatment of otalgia.

The glossopharyngeal nerve is also associated with a variety of pain syndromes and therefore is a surgical target. The glossopharyngeal nerve receives fibers from the inferior salivatory nucleus, nucleus ambiguus, nucleus of the solitary tract, and spinal trigeminal nucleus. It emerges from the medulla, and its sensory functions include sensation to the pharynx, soft palate, posterior tongue, tonsils, and ear (including the tympanic membrane, eustachian tube, and some components of the outer ear and ear canal). Fibers of the glossopharyngeal nerve pass through the superior ganglion just proximal to the foramen magnum, and the inferior ganglion distal to the foramen magnum, where it begins to branch.²

The first branch of the glossopharyngeal nerve, the tympanic nerve, carries sensory fibers (spinal nucleus of trigeminal nerve) from the tympanic cavity and eustachian tubes. The glossopharyngeal nerve also supplies branches to the carotid sinus, pharyngeal plexus, the stylopharyngeal muscle, and the pharyngeal tonsils and posterior one third of the tongue.

Cranial nerve X may itself be implicated in a variety of pain syndromes. The vagus nerve is composed of fibers from several brainstem nuclei: dorsal nucleus of the vagus, nucleus ambiguus, nucleus of the solitary tract, as well as the spinal trigeminal nucleus.² The vagus nerve exits the brainstem at the level of the medulla, passes through a superior ganglion proximal to the jugular foramen and an inferior ganglion distal to the foramen magnum. The vagus has a number of branch points in the neck, including pharyngeal branches, the superior laryngeal nerve, recurrent laryngeal nerve, and cervical cardiac branches. The sensory role of the vagus includes transmitting sensory information from the dura of the posterior fossa, from the external auditory canal, and from a region posterior to the auricle. Sensory information is also transmitted from the lower pharynx and the larynx, from receptors in the aortic arch and aortic body, and from visceral organs.²

Both the glossopharyngeal nerve and the vagus nerve exit the skull via the jugular foramen. The vascular structures passing through the foramen—the jugular vein, inferior petrosal sinus, and the posterior meningeal artery—are in general posterior to the neural structures passing through the foramen. The spinal accessory nerve is the most lateral and posterior of the cranial nerves as they enter the foramen, and

is just antromedial to the posterior meningeal artery. The vagus nerve is antromedial to CN XI, and the glossopharyngeal nerve is antromedial to the vagus, traversing through the most anterior aspect of the foramen.² Often, the glossopharyngeal nerve is separated from the other two nerves by a fibrous or bony band.

The pterygopalatine, or sphenopalatine, ganglion as well as the vidian nerve (nerve of the pterygoid canal) may be implicated in one of several pain disorders. The vidian nerve is formed where the greater superficial petrosal nerve joins the deep petrosal nerve. It traverses the pterygoid canal to enter the pterygopalatine ganglion.³ The ganglion itself acts as a relay station for a number of motor, sensory, and autonomic pathways.³ The ganglion is located in the pterygopalatine fossa, which is inferior to the sphenoid sinus, anterior to the medial plate of the pterygoid process, and posterior to the maxillary sinus and middle turbinate.³ There are six routes via which neurovascular structures enter the fossa: foramen rotundum, pterygoid canal, greater palatine canal, lesser palatine canals, sphenopalatine foramen, and the inferior orbital fissure.² Sensory fibers originate from the maxillary nerve and supply sensation to the nose, throat, and sinuses.³ Synapsing parasympathetic fibers originate with CN VII/nervus intermedius, pass through the geniculate ganglion and ultimately reach the pterygopalatine ganglion via the greater superficial petrosal nerve-come-vidian nerve; sympathetic fibers from the internal carotid plexus join the deep petrosal nerve-come-vidian nerve, pass through the ganglion without synapsing, and join the maxillary nerve.^{2,3}

■ Pain Syndromes and Surgical Approaches

Chronic Neurogenic Pain, Central Pain, and Poststroke Pain

Syndromes

Chronic neurogenic pain is a condition that may arise from a lesion along the pain system axis, from abnormal signaling in the absence of a clear lesion, or after the removal of the inciting lesion. The International Association for the Study of Pain Task Force on Taxonomy has defined central pain as “pain due to a lesion or dysfunction of the central nervous system.”⁴ According to an article by Nurmikko in 2000, a significant number of stroke patients (8%), spinal cord injury patients (50%), as well as syringomyelia/syringobulbia (80%) patients complain of some degree of pain.⁵ Furthermore, poststroke pain often affects the thalamus (61%), most often the ventroposterior region, but 39% of cases do not demonstrate clear thalamic injury.⁵ Inversely, stroke may place the patient at decreased risk of central pain, as in the case of brainstem stroke involving the bilateral quintothalamic tracts.⁴ Studies suggest that dysfunction of the spinothalamocortical tract is a necessary but in some cases not a sufficient cause of central pain.⁴

Although stroke is believed to be the most common cause of central pain, this is most likely due to the fact that stroke is much more common than other etiologies, rather than possessing a greater predilection to cause central pain.⁴ Central pain itself may present in a variety of different ways and involve disparate locations. Most commonly, poststroke pain is described as having a cold, burning quality, and there is often (72%) a component of allodynia.⁴ Stroke patients may also describe novel sensations which, although not painful, are exceedingly

unpleasant.⁴ In contrast, patients with spinal cord involvement often describe dysesthesia, or “pins and needles” sensations.⁴ Multiple sclerosis (MS) patients may describe aching pain, stabbing pain, or burning sensations.^{4,5} It should be noted that although some causes of central pain are of brainstem origin and may mimic CN neuralgias, central pain often manifests with other signs or symptoms of brainstem dysfunction.

Central pain has been hypothesized to occur due to deafferentation and resultant overactivity of neural elements upstream from an insult, or to damage and subsequent disruption of previously homeostatic signaling pathways.⁴ Nurmikko points out that these two hypotheses assume that higher levels of processing and perception haven't been disrupted, either directly via insult or indirectly via alterations in downstream circuitry; nor do they account for the neuropsychiatric changes that may arise amid a maelstrom of chronic suffering.⁴

In a series of publications, Jeanmonod et al demonstrated the presence of abnormal calcium spike bursts that they hypothesized result in “self-perpetuating thalamic cell membrane hyperpolarization, similar to the one seen in slow wave sleep.”⁶ They go on to hypothesize that this hyperpolarization of thalamic cells may be responsible for other positive symptoms following central insult, such as “tinnitus, abnormal movement, epilepsy and certain neuropsychiatric disorders.”⁶ In another report by the same group, they describe microelectrode analysis of the medial thalamus in 45 patients undergoing medial thalamotomy for pain, and found that the most effective therapeutic results correlated to regions in and around the central lateral nucleus demonstrating low-threshold calcium spike bursts. They hypothesize that central pain involves an imbalance and overinhibition of central lateral and ventroposterior nuclei by the reticular nucleus.⁷ The hypothesis states that after a lesion occurs involving the lateral spinothalamic tracts, there is a preferential decreased excitation of VP as compared with CL. The spared excitation of CL results in overactivation of the reticular nucleus and subsequent inhibition of VP. This inhibition causes low-threshold spikes, activation of the reticular nucleus, and inhibition and subsequent generation of low-threshold spikes involving CL, giving rise to a positive feedback loop⁷ (**Table 18.1**).

Surgical Approaches

Deep brain stimulation (DBS) has been used for the management of chronic pain as well as phantom limb pain. The traditional target has been the periaqueductal gray/periventricular gray region, although the mechanism of action remains unknown.⁸ Cortical stimulation is another modality that has been used to treat a variety of pain disorders, from poststroke pain⁹ to cranial neuralgias.

Radiofrequency ablation has been used for many years to target the thalamus in the treatment of neuropathic pain.¹⁰ Central pain was first hypothesized in 1911 by Head and Holmes, but the ability to perform medial thalamotomies for central pain was realized with the emergence of stereotactic techniques in the 1950s.¹¹ The procedure was initially embraced as low risk and with minimal side effects, but symptomatic recurrence was frequent.¹¹ Classically, caudal regions in the intralaminar complex, such as the centromedian/parafascicular complex, central lateral nucleus, posterior complex, and medial pulvinar, were targeted.¹¹ In the *Textbook of Stereotactic and Functional Neurosurgery*, Jeanmonod and Morel discuss targeting of the posterior central lateral nucleus “based on recent multi-

Table 18.1 Surgical options for nontrigeminal pain

Etiological site	Pain location	Surgical approaches
Central pain/poststroke ^{8,9,11–13}	Throughout the body	1. Medial thalamotomy—radiofrequency 2. Medial thalamotomy—radiosurgical 3. Medial thalamotomy—focused U/S (experimental) 4. Cortical stimulation 5. Periaqueductal gray DBS 6. Focused U/S
Vagoglossopharyngeal ^{12,14,16–18}	Lancinating pain in posterior oropharynx, base of the tongue, below the angle of the jaw; may involve the ear; may be accompanied by hemodynamic instability/syncope	1. Microvascular decompression 2. Case reports of epidural sensory cortex stimulation 3. Gamma Knife
Geniculate/nervus intermedius ^{19–22}	Lancinating paroxysmal pain within the ear	1. Microvascular decompression 2. Nerve sectioning
Pterygopalatine neuralgia (Sluder syndrome) ^{3,19,23,25}	Retro-orbital, zygomatic, nasal pain; may have numbness of the nose, soft palate, pharynx; parasympathetic overactivity	1. Blocks 2. RF stimulation 3. RF ablation
Vidian neuralgia (Vail syndrome) ²⁵	Variant of Sluder. Pain may radiate into the shoulder, neck, or ear, and may be accompanied by vertigo or tinnitus	Block
Cluster headache ^{13,18,23,24,26}	Paroxysmal unilateral attacks centered on temple, cheek, eye; accompanied by autonomic dysfunction	1. Pterygopalatine blocks or stimulation 2. DBS of posterior hypothalamus 3. RF ablation of pterygopalatine ganglion
Superior laryngeal neuralgia ²⁷	Unilateral paroxysmal pain centered around hyoid bone, thyroid cartilage; may radiate to jaw or ear	Block

Abbreviations: DBS, deep brain stimulation; RF, radiofrequency; U/S, ultrasound.

architectonic studies [due to integration of] the nucleus in a large thalamocortical network responsible for the multiple sensory, cognitive, and affective components of the neuropathic pain condition.”¹¹

In advocating the targeting of the central lateral nucleus, Jeanmonod and Morel make four points: (1) afferents from the spinothalamic tract to the nucleus are known, in contrast to the lack of data establishing afferents to the central median/parafascicular complex or medial pulvinar; (2) fibers from layer I and layers III–IV passing through the central lateral nucleus project to regions governing a wider array of pain processing than other targets, such as the “discriminative (SI, SII), affective-motivational (ACC, insula), cognitive (PFC), and motor (motor cortex)”¹¹; (3) the central lateral nucleus is farther from the primary somatosensory nuclei, reducing the risk of sensory deficits as compared with CM lesioning; (4) there is low anatomic variability in this target.¹¹

The procedure itself is performed using a magnetic resonance imaging (MRI)-compatible frame, and under local anesthetic. Localization is achieved via stereotactic MRI. Axial images parallel to the intercommissural plane are derived. The lead is placed with the assistance of impedance monitoring in the posterior portion of the central lateral nucleus via a prefrontal approach, anterior to the coronal suture. The target is located at the level of the intercommissural plane, 2 mm posterior to the posterior commissure, and 6 mm lateral to the thalamo-ventricular border.¹¹ A lesion 1 to 2 mm above the intercommissural plane may be made to avoid lesioning the pretectum. The medial-lateral angle is between 5 and 10 degrees, and the anteroposterior (AP) angle between 60 and 65 degrees. This AP angle may be less than 60 degrees in some patients, necessitating a 1 mm posterior correction in some cases. Partial lesioning of the CL is adequate in most cases.¹¹

Microelectrode recording is performed while the patient is asked to carry out various motor tasks. Additionally, tactile, nociceptive, and proprioceptive stimuli are applied and monitored. Once recording is complete, the microelectrode is replaced with a blunt pencil tip macroelectrode, and stimulation is applied to assess for any motor, cognitive/emotional, or sensory responses. Most patients experience paresthesias or dysesthesias localized to the affected region, but may experience full-body responses. Radiofrequency lesioning of 10 to 12 mm length by 4 mm diameter is then performed.¹¹

Ninety-six patients between the ages of 18 and 84 with chronic, medically refractory central or peripheral pain underwent lesioning in the study. Over 60% of the patients had undergone previous transcutaneous electrical nerve stimulation (TENS), dorsal column or thalamic stimulation, or ablative procedures such as sympathectomy, neurotomy, cordotomy, or rhizotomy.¹¹ Follow-up was from 2 weeks to over 10 years. Close to 20% of patients experienced complete pain relief, whereas 53% demonstrated over 50% pain relief. Paroxysms were reduced by 65% and the duration of pain episodes was reduced by 90%.¹¹

Radiosurgical ablation offers a less invasive technique for performing medial thalamotomy. Frighetto et al propose linear accelerator thalamotomy as an alternative to more invasive techniques. In the series of three patients, two presented with poststroke pain and the third with malignant pain secondary to invasion of the brachial plexus.¹² In the study, 150 to 200 Gy were administered to the target for 55 to 85 minutes using a 5-mm collimator. A BRW frame was first fitted parallel to the AP commissure plane.⁹ The centromedian-parafascicular nuclei were targeted using MRI and the Schaltenbrand and Warren atlas, where the atlas was

sized to the patient's MRI at the AP commissure plane.⁹ Patient 1 was treated for refractory pain following an MCA stroke and demonstrated immediate pain relief that lasted 4 months.⁹ She subsequently required motor cortex stimulation for refractory pain. Patient 2 presented with small cell carcinoma with invasion of the brachial plexus. He had previously deferred brachial plexus ablation. Due to comorbidities and a trial of medically induced coma for pain relief, it was felt that stereotactic thalamotomy was the best and safest option. He was able to return home following the procedure with good pain relief and decreased requirement of pain medication.⁹ Patient 3 presented with refractory pain of the face, hand, and foot following a right posterior cerebral artery infarct with right thalamic lacunar infarct. She demonstrated an immediate decreased pain medication requirement and decreased allodynia, and at 3 years was taking pain medication only twice a week.⁹

Ultrasound therapy is an older modality, which is nonetheless experiencing a re-emergence as localization technology becomes more sophisticated. Jeanmonod et al reported the use of MRI-guided focused ultrasound for central lateral thalamotomy. The procedure was performed on 12 patients with a variety of medically refractory neuropathic pain disorders, including postherpetic neuralgia, traumatic trigeminal nerve injury, lumbar radiculopathy, thalamic infarct, and brachial plexus avulsion.⁹ Using 3T MRI-guided focused ultrasound via the ExAblate 4000 (InSightec, Haifa, Israel), the authors completely shaved the patient's head, covered it with a special fenestrated silicone membrane, and placed the patient's head into an MRI-compatible stereotactic frame. The silicone membrane was used to circulate water at 15 to 20°C around the head. MR imaging was obtained and the target—the posterior portion of the thalamic central lateral nucleus—was identified. The target was then heated to between 39 and 42 degrees for 10 to 20 seconds with sublesioning pulses. This allowed the targeted tissue to be confirmed with MR thermography. Once confirmation was achieved, a series of higher sonifications were applied, with a target tissue temperature of 51 to 64°C. The authors noted that although thermo-coagulation effects may be seen at a temperature of 50°C, tissues must reach 55 to 57°C to ensure a 100% lesioning effect.¹³ Sonications were of 10 to 20 seconds in duration, with up to 12,000 J per sonication.¹³ Mean pain relief at 3 months was 47% (of 9 patients) and 57% at 1 year (8 patients). The potential for ultrasound therapy to supplement radiofrequency ablation or radiosurgical ablation seems promising, particularly in those patients for whom a more invasive procedure is prohibitive. However, more work needs to be done in this arena before the technique can enjoy a wider acceptance (**Table 18.1**).

Glossopharyngeal Neuralgia

Anatomy and Syndrome

Chronic pain involving the cranial nerves may be due to tumor compression, compression from normal or abnormal vascular structures, postinfectious chronic nerve irritation, or demyelinating disorders. Glossopharyngeal neuralgia (or vagoglossopharyngeal neuralgia, as some have proposed^{14,15}) describes a condition where the patient experiences paroxysmal attacks of lancinating pain in the posterior oropharynx, the base of the tongue, beneath the angle of the jaw, and possibly the ear.^{14,15} Attacks may be triggered by oropharyngeal maneuvers,

raising the arm, or turning the head, and may be accompanied by hemodynamic instability resulting in syncopal episodes (suggesting vagal involvement).¹⁶ Most cases present between the ages of 40 and 60, and often involve the left side. It is rare; reported incidence varies among studies, but has been cited as between 0.2 and 0.7 per 100,000 per year,¹⁴ with slight predominance of men at 0.9 per 100,000 per year versus women at 0.5 per 100,000 per year.¹⁵ It has been noted that 10% of cases copresent with trigeminal neuralgia.¹¹

Glossopharyngeal neuralgia may be subdivided into pharyngeal, otalgic, and vagal neuralgias, depending upon the predominant constellation of symptoms.¹⁴ In a case series of 19 consecutive patients treated for glossopharyngeal neuralgia, Gaul et al found that although 3 patients presented with concomitant vagal neuralgia, all patients were found to have vascular compression of the vagus nerve at the time of surgery.¹⁴ Compression of the nerve by posterior inferior cerebellar artery (PICA) was the most common finding at the time of surgery, although compression by PICA and the vertebral artery, or the anterior inferior cerebellar artery (AICA) and the vertebral artery was also observed.¹⁴ In a series of 31 patients, Ferroli et al found that the vertebral artery or the AICA alone could act as an offending vessel, and documented 1 case where the offending vessel was a vein¹⁶ (**Table 18.1**).

Surgical Interventions

Microvascular decompression (MVD) has been successfully used to treat trigeminal neuralgia for many years. Dandy was the first to note compression of the trigeminal nerve by a vascular loop in 1934,¹⁶ and 2 years later Lillie and Craig reported on a patient with glossopharyngeal neuralgia (GN) thought to be caused by a compressive vessel loop.¹⁶ But it wasn't until the 1960s that microvascular decompression for cranial neuralgias was articulated and popularized by Dr. Jannetta.¹⁶ Given the rarity of glossopharyngeal neuralgia, as well as the hazards of operating upon the lower cranial nerves, there are scant long-term outcome studies of patients undergoing MVD for GN.

In a study published in 2009, Ferroli et al looked at 31 patients over 18 years (1990–2007) who underwent MVD for GN.¹⁶ Surgical technique involves exploration of the cerebellopontine angle via a retromastoid approach. In this particular approach, the authors identified the “margin of the sigmoid sinus from its beginning to the region behind the mastoid tip.”¹⁶ The dura was incised parallel to the margin of the sigmoid, and the cerebellomedullary cistern was opened to allow for access to the lower cranial nerves as well as for cerebrospinal fluid (CSF) drainage.¹⁶ The authors describe use of the endoscope in 4 of the cases to fully visualize the root entry zones of cranial nerves IX and X.¹⁶ Compressive vessels were identified and microvascular decompression accomplished using Teflon (3M, Minneapolis, MN, USA) or muscle fragments of fibrillar Surgicel (Ethicon, Cincinnati, OH, USA).¹⁶ If venous structures were thought to be the etiological culprit, these were coagulated and cut. In their series, 18 cases were left sided, and the remaining right sided. Twenty-two cases demonstrated compression by the PICA, 7 due to vertebral artery compression, and 1 each due to AICA and a compressive vein.¹⁶ Immediate pain relief was noted in 28 cases, with the other 3 patients experiencing gradual relief over the span of a few weeks.¹⁶ Two patients required reoperation for medically refractory recurrence, and were found to have incomplete decompressions at the time of surgery.¹⁶

In 2011, Gaul et al published a series of 19 consecutive patients (18 of whom underwent surgery, and 1 of whom was excluded due to medical control of pain) from 1994 to 2009 referred for refractory glossopharyngeal neuralgia. Patients were subdivided into vagal, pharyngeal, and otalgic subtypes based upon clinical presentation. All patients underwent preoperative MRI with 3D (three-dimensional) reconstructions. In this series, the authors used a retrosigmoid approach with extension into the foramen magnum. The cerebellopontine cistern was opened for CSF drainage, and cranial nerves IX and X were identified. Offending vessels were dissected free and isolated using Teflon.¹⁴ Twelve cases were left sided and 7 right sided. Although all patients presented with pharyngeal pain, 12 patients demonstrated additional otalgic symptoms, and 3 suffered vagal symptoms.¹⁴ Of the 18 surgical patients, 16 were pain free and 2 demonstrated a decrease in their pain.¹⁴

In the rare case that a patient is refractory to medical and conventional surgical management, there are other options. A 2010 case report by Anderson et al described cortical stimulation in a patient with medically and surgically refractory glossopharyngeal neuralgia, trigeminal neuralgia, and dysphagia.¹⁷ Signs and symptoms included left-sided facial pain, throat pain, nausea/vomiting, and dysphagia due to the pain. She had previously undergone multiple open and percutaneous procedures, to no avail. The patient underwent an initial awake craniotomy for placement of an epidural trial electrode across the central sulcus. The electrode position was then adjusted to elicit lower facial and tongue responses using a pulse generator set at 5 Hz and 250 μ s. Once the desired location was found, the electrode was attached to the dura. After the trial week, in which the patient's pain went from 10/10 to 6 to 7/10 with stimulation, the lead was tunneled and attached to a subclavicular generator. In the postoperative period, the patient required adjustments and generator replacement, but experienced pain relief as well as decreased dysphagia and nausea/vomiting.¹⁷

For patients who are not good surgical candidates, or have refused open surgery, radiosurgical intervention is an option. In a 2010 report, Williams et al described the case of a 47-year-old woman who presented with medically refractory, lancinating left throat pain.¹⁸ She declined MVD, so was offered GK therapy. A dosage of 80 Gy was administered to the nerve as well as the glossopharyngeal meatus, and the patient remained neurologically intact and pain free at 12 months' follow-up¹² (**Table 18.1**).

■ Geniculate/Nervus Intermedius Neuralgia Syndrome

Geniculate neuralgia (nervus intermedius) describes a condition where patients experience lancinating, paroxysmal pain within the ear. Pressure upon the tragus, sound, cold, or swallowing may trigger pain.¹⁹ The diagnosis is one of exclusion; that is, a full otolaryngological examination must be performed, including vestibular examination; audiogram; evoked potentials; full examination of the mouth, nose, nasopharynx, pharynx, larynx, and paranasal sinuses; as well as MRI of the brain, to rule out other etiologies.²⁰ Because there is some overlap in the sensory

distribution of cranial nerves V, VII, IX, and X, diagnosis may be difficult, or may point to involvement of two or more nerves¹⁹ (**Table 18.1**).

Surgical Interventions

Nervus intermedius (NI) neuralgia has classically been treated with nerve sectioning. Using a standard retrosigmoid approach, the nerve is identified and dissected as it exits the brainstem between CNs VII and VIII, and it may be sectioned using standard techniques. A 2005 article published by Ashram et al described intraoperative electrophysiologic identification of the NI.²¹ Although this report describes identification in the context of various CP angle procedures, it could prove useful for procedures specifically targeting NI neuralgia. The study was a retrospective case review of 33 patients who underwent facial nerve monitoring using electrodes placed in the orbicularis oculi and orbicularis oris. They found that stimulation produced a long-latency, low-amplitude response in the orbicularis oris electrode at a mean threshold of 0.4 V, a mean latency of 11.1 ms, and a mean amplitude of 11.1 mV.²¹ These responses were found to be distinct from facial nerve responses.²¹

Microvascular decompression has also been proposed as a treatment for otalgia, or NI neuralgia. In a case report published in 2011 in the *Journal of Laryngology and Otology*, Saers et al report upon a 24-year-old with a 9-year history of otalgia.²² The patient presented with a deep lancinating pain within the left ear refractory to a variety of medications and blocks. Hearing and cranial nerve function were preserved. Imaging demonstrated anomalous AICA between the facial and vestibulocochlear nerves, in the region of the NI.²² Surgical exploration was performed, and several blood vessels as well as arachnoid adhesions were noted overlying the facial and vestibulocochlear nerves.²² The adhesions were removed and the vessels were carefully dissected away and isolated from the nerve complex using Gelita-Spon (Gelita Medical, Eberbach, Germany) and Tissucol Duo 500 (Baxter, Vienna, Austria).²² The patient awoke without deficit and remains pain free. Whereas sectioning of the NI remains a popular option for NI neuralgia, MVD may be considered in certain cases (**Table 18.1**).

■ Pterygopalatine/Sphenopalatine Neuralgia, Vidian Neuralgia, and Cluster Headaches Syndromes

Pterygopalatine/sphenopalatine ganglion neuralgia, or Sluder syndrome, is a particular pain disorder that manifests with gustatory, motor, and sensory abnormalities in addition to pain,³ which may be due to previous inflammation/infection of the posterior ethmoid and sphenoid sinuses.¹⁹ The condition may involve paroxysmal attacks of parasympathic overactivity, causing tearing, nasal congestion, eye infection, and alteration in taste along with retro-orbital, zygomatic, and nasal pain, and accompanied by numbness of the nose, soft palate, and pharynx.³ There is some speculation that the sphenopalatine ganglion is involved in cluster headaches, and therefore it has been proposed as a target in the treatment of cluster headaches.^{23,24}

Vidian neuralgia, or Vail syndrome, may be considered a variant of sphenopalatine neuralgia, and occurs when the nerve is irritated in the pterygoid ca-

nal due to trauma or inflammation/infection. It is paroxysmal and unilateral, with pain radiating into the shoulder, neck, or ear, and may be accompanied by vertigo and tinnitus.²⁵

The term *cluster headache* describes paroxysmal unilateral attacks centered around the temple, cheek, or eye that are accompanied by autonomic dysfunction such as ocular swelling, tearing, and nasal congestion.²⁶ Although the etiology is poorly understood, a variety of targets have been proposed for therapy, including the posterior hypothalamus and sphenopalatine ganglion.^{23,26} Cluster headaches may occur in bursts for up to 12 weeks followed by periods of remission, or may be chronic in up to 10% of sufferers, occurring for a year or more without remission.²⁶ There are known triggers, such as bright lights and alcohol ingestion, but studies have demonstrated a circadian relationship as well, suggesting an endocrinologic role in the generation of cluster headaches.²⁶ Prevalence has been quoted as between 0.006 % and 0.24%, depending upon geographic location²⁶ (**Table 18.1**).

Surgical Interventions

Sphenopalatine blocks may be performed using the fluoroscopic-guided percutaneous technique described below. A needle is advanced inferior to the zygomatic arch, advanced through the coronoid notch onto the pterygoid plate, and anteriorly along the plate and into the fossa.²³ Anesthetic may then be injected into the ganglion.

Vidian neuralgia may be treated with a block, by entering the pterygoid canal via the greater palatine foramen and advancing the needle into the canal. Local anesthetic may be instilled or neurolysis performed with alcohol.

Both radiofrequency ablation and radiosurgery have been proposed as treatments for sphenopalatine neuralgia and cluster headaches, by targeting the sphenopalatine ganglion or the trigeminal nerve. A 2007 article by Lad et al followed a patient with medically refractory cluster headaches who had undergone two successful block trials prior to undergoing radiosurgery. A single fraction of 45.5 Gy was administered to the 78% isodose line to a maximum dose of 65 Gy.²⁴ At 12-month follow-up, the patient demonstrated a decrease in the number and severity of attacks, as well as a decrease in medication usage.

Radiofrequency ablation of the sphenopalatine ganglion involves an infrazygomatic arch approach. Ruiz-Lopez et al describe a technique where the clivus, sella, petrous bone, and pterygopalatine fossa are identified on a lateral film.²⁵ The needle is inserted perpendicular to the skin in the upper region of the mandibular arch and advanced to the fossa until the patient experiences paresthesias in the jaw.²⁵ An AP film is then used to advance the needle medially, 1 to 2 mm above the vomer, until the tip is adjacent to the lateral wall of the nasal cavity.²⁵ Upon stimulation, paresthesias should occur in the nasal region and the palate, and no maxillary contraction should be observed.²⁵ If the paresthesias are noted in the palate only, the electrode should be slightly advanced.²⁵ Inject 1 ml of 2 percent lidocaine prior to lesioning. Single lesioning may be performed via pulsed radiofrequency for 4 minutes.²⁵ Pulsed RF may also be used to create 3 lesions—one in the PPF, one located 1–2 mm medially, and one located 3 mm medially—though the duration of each is 1 minute rather than 4.25. Alternately, conventional RF at 80°C may be used to create the above three lesions (PPF, 1–2 mm medial, and 3 mm medial), with a duration of 1 minute for each lesion.²⁵

Radiosurgical targeting of the trigeminal nerve has also been proposed as a treatment for cluster headaches, although a 2006 study by McClelland et al does not bear this out. The study followed 10 patients who had undergone Gamma Knife (Elekta, Stockholm, Sweden) surgery of the trigeminal nerve. They found poor pain relief in 9 patients immediately following radiosurgery.¹³ Although 6 patients improved between 2 weeks and 2 years, they ultimately regressed. Half of the patients experienced facial numbness following the procedure.¹⁸

In 2010 Ansarinia et al published a paper targeting the sphenopalatine ganglion (SPG) for the treatment of cluster headaches via radiofrequency stimulation. The authors followed 6 patients treated with up to 1 hour of sphenopalatine stimulation during a cluster headache attack. These attacks were spontaneous or were triggered using pungent smells, bright lights, IV nitroglycerin, or alcohol ingestion.²³ The treating physicians used a fluoroscopically guided percutaneous infrazygomatic transcoronoid approach, placing the electrode into the pterygopalatine fossa, adjacent to the sphenopalatine ganglion.²³ A 20-gauge foramen needle was used to enter inferior to the zygomatic arch and advance through the coronoid notch and onto the pterygoid plate, where it was then advanced anteriorly into the pterygopalatine fossa.²³ Next, a single-contact temporary electrode was placed into the needle and advanced toward the ganglion. Paresthesias in the root of the nose and posterior nasopharynx were induced via stimulation of various intensities (less than 2 V) at 50 Hz and 300 μ s to confirm physiological placement of the electrode.²³ Treatment was initiated when patients rated their pain as 8 (out of 10) or greater, and stimulation parameters were adjusted so as to effect pain relief. When the cluster headache resolved it was reinduced with known triggers and allowed to reach its maximum intensity prior to restimulation.²³ This cycle was repeated for a total of 1 hour per patient.²³ Of the 18 attacks investigated in the study, 61% (11 of 18) demonstrated full resolution, whereas 4 attacks demonstrated no resolution and 3 demonstrated partial resolution.²³ Autonomic features such as ocular swelling and nasal congestion also demonstrated resolution with stimulation.²³ The authors concluded that the SPG may offer a reasonable target for permanent neuromodulation in the treatment of cluster headaches, although the investigation is ongoing.

In 2002, Franzini et al published a study targeting the posterior hypothalamus for the treatment of cluster headaches. The study followed 5 patients who had undergone long-term, high-frequency stimulation via implantable electrodes. Electrodes were placed 2 mm lateral to midline, and 3 mm posterior and 5 mm inferior to the midcommissural point.²⁶ At 2 to 22 months follow-up period, all patients were pain free, 2 patients were pain and medication free, while 3 required continued low-dose medication (methysergide and verapamil).²⁶

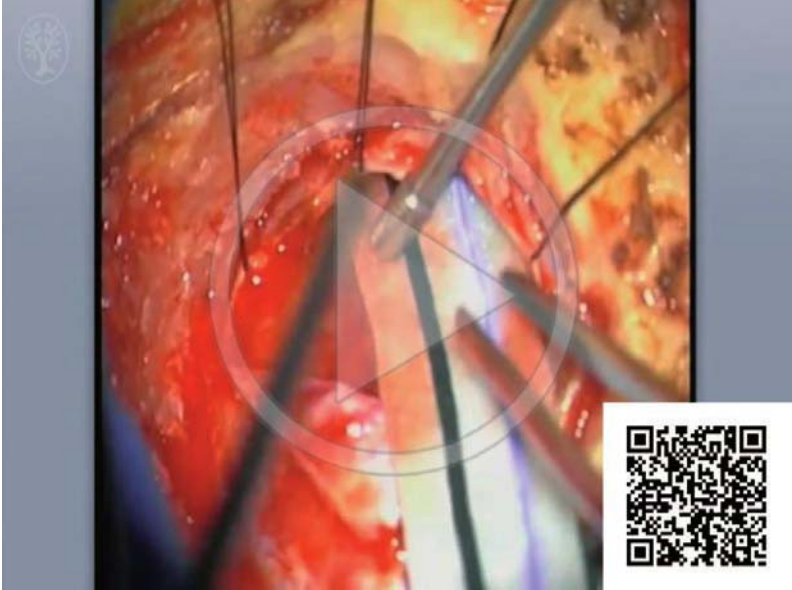
Electrodes were implanted using the Leksell stereotactic frame and under local anesthetic and/or low-dose benzodiazepine or propofol.²⁶ High-resolution MRI was used to determine the anterior commissure–posterior commissure line, and was fused with a computed tomography (CT) scan obtained under stereotactic conditions.²⁶ A precoronal paramedian bur hole was used to advance a cannula up to 10 mm from the target.²⁶ The microrecording electrode followed by the definitive electrodes were advanced to the target. Macrostimulation was used to elicit potential side effects, and adjustments were made until these were minimized. After a trial period of 7 to 10 days, the leads were connected to a subclavicular generator (**Table 18.1**).

■ Superior Laryngeal Neuralgia Syndrome

Superior laryngeal neuralgia is a very rare disorder that may manifest with unilateral paroxysmal pain centered around the hyoid bone or thyroid cartilage, although it may radiate to the jaw or ear.²⁷ Other causes for pain, such as mass lesion, must be ruled out with imaging and full ENT examination (**Table 18.1**).

Surgical Intervention

Superior laryngeal nerve blocks may be performed by injecting high-dose local anesthetic into the region where the nerve enters the hyothyroid membrane.²⁷ The block tends to be short acting, although in an article published by Sato et al, they reported long-term pain relief over the course of many sessions using high-dose lidocaine.²⁷ Care must be taken to avoid injection of anesthetic into the superior thyroid artery.²⁷ Of benefit, this procedure avoided the side effects of neurolysis with alcohol or phenol²⁷ (**Table 18.1**).



Video 2. Nervus Intermedius Transection for Geniculate Neuralgia

Editor's Comments

The host of diagnoses that are included here make this chapter a difficult assignment, both for the authors and the reader. Whereas trigeminal neuralgia is a relatively common and reasonably definable entity, a listing of other, more uncommon cranial neuralgias quickly becomes somewhat obscure. I congratulate Drs. Barbaro and Hill for producing this readable review.

Given the rarity of these conditions, careful prospective series with well-described outcomes are a rarity. In some instances, even the diagnoses are controversial. For example, there is not a consensus concerning the distinction between sphenopalatine neuralgia and cluster headache. Other conditions such as vidian neuralgia and superior laryngeal neuralgia are so obscure and indeterminate that their mere existence may be supported solely by comprehensive lists of historical diagnoses in chapters such as this. In these instances, it is certainly possible that these "named" diagnoses may simply be unusual variants of more common conditions, referable to their anatomical region.

What we can glean from the evidence and the published experience is that there does seem to be a distinct entity that we call glossopharyngeal neuralgia (GPN). This condition refers to episodic lancinating or stabbing pain in the tonsillar region or the posterior tongue. Pain can be referred to the throat just behind the angle of the jaw. It is usually triggerable by swallowing. In the past it was referred to as vagoglossopharyngeal neuralgia because sufferers could experience syncope, apparently due to bradycardia and hypotension from vagal nerve activation. In my experience, this accompaniment to the pain is rare to nonexistent.

The evidence to support a surgical approach to a patient with medically intractable GPN is based on case series, but these results do comport with my experience, as well. Using high-resolution MRI and MRA, we have found that essentially every patient with this clinical diagnosis has demonstrable vascular compression of cranial nerves IX and X, usually from the posterior inferior cerebellar artery (PICA), sometimes from the anterior inferior cerebellar artery (AICA), and rarely from the vertebrobasilar complex. Microvascular approach to these nerves is the most effective strategy, particularly when vascular decompression is combined with complete section of the glossopharyngeal nerve and partial section of the vagus nerve (section of the most superior one or two rootlets).

In my opinion, geniculate neuralgia (intermedius neuralgia) may or may not be a legitimate syndrome. Because it is so rare, it is difficult for any one surgeon to mount much of an experience base. When a patient tells me that he has paroxysmal stabbing pain deep in the ear only ("an ice pick in the ear"), this diagnosis comes to mind. The diagnosis is based solely on the patient's complaint because, at least in our center, imaging has not yielded any consistent results. I have personally operated on around a half dozen patients in whom I thought the diagnosis applied. I have performed section of the one or two rootlets of the nervus intermedius. This can be a somewhat daunting task; these rootlets lie between cranial nerves VII and VIII and potential complications can include facial weakness and hearing loss. In my small case series, the results have been mostly gratifying, but I am still unsettled as to the nature and diagnosis of this syndrome.

The evidence to support radiosurgical treatment or radiofrequency ablation of the sphenopalatine ganglion for sphenopalatine neuralgia or cluster headache is simply too meager to recommend these options at this time. Other treatments for neuropathic pain, such as deep brain stimulation and medial thalamotomy, are discussed in other chapters. These are not potential treatments exclusively for cranial neuralgias, and the data to support them must be comprehensively evaluated for each diagnosis in which they are applied.

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Percutaneous Computed Tomography–Guided Trigeminal Tractotomy and Nucleotomy

Yücel Kanpolat

The pain sensation reaches the central nervous system via a transmission system. Therefore, surgical destruction of this transmission pathway has been considered as a treatment method in pain surgery. Concepts regarding destruction of the pain transmission system emerged as a result of clinical observations based on the destruction of this system due primarily to accidents, injury, trauma, or tumor.¹ Cordotomy emerged as a result of such a coincidental observation, and was suggested by a meticulous neurologist, Spiller, before the functional structure of the lateral spinothalamic tract had been described.^{2–4}

Stereotaxic destruction of the pain-transmitting system is the basis of stereotaxic pain procedures. Stereotaxy can be described as a technique that utilizes a three-dimensional coordinate system to locate a specific region in the body with the reference of instruments such as needles or electrodes.⁵ In my experience, stereotactic pain surgery is performed in three stages: (1) direct morphological localization of the target point with stereotaxic methods, which can be performed with computed tomography (CT) or magnetic resonance imaging (MRI) in today's facilities; (2) measurement of the resistance of the target point using the impedance method and confirmation of the functional status with stimulation; and (3) controlled, selective destruction of the target point or tissue. This can be done with radiofrequency (RF) energy of a specific size or temperature. Operations in which these three basic principles are applied can be referred to as stereotaxic. These applications are surgeries in essence, and they should be performed by an experienced neurosurgeon.

The stereotaxic pain procedures, which require specific knowledge and experience, have had quite encouraging outcomes. Unfortunately, these procedures, which are applied to relieve intractable pain mostly in cancer patients, have been prevented from attaining popular use due to the complication and mortality rates reflected in data from obsolete literature.⁶ Today the majority of patients experience difficulty in finding a physician who can relieve their pain. According to a study in the United States, one in four patients has had to change physicians at least three times because of persistent pain. This is also a reflection of the generally insufficient knowledge and experience of physicians in the field of pain management.⁷

Today fundamental procedures in pain surgery have been developed by neurosurgeons, and these valuable and important techniques are being proffered on golden trays to other medical occupational groups. We live in a time commonly accepted as the science and information age. In my opinion, physicians in this century are guided by company dogmas. The use in developed Western countries of numerous procedures that are of no medical value supports this

opinion. Unfortunately, today the benefit of some procedures is extremely debatable but their efficacy or popularity is not. A patient's sleep quality, pain-free status, appetite, and defecation habits are important parameters in the practice of pain management, especially in cancer patients, and shape their quality of life. Many physicians today use arguable algorithms and scoring systems rather than evaluate these fundamental parameters. Today, using the analgesic ladder recommended by the World Health Organization (WHO) in the late 1980s⁸ as a gold standard is inappropriate with respect to treatment realities. Despite the reliance on guidelines, persistent pain problems have been identified at rates of up to 40% in some studies.^{9,10} Cancer pain is usually managed at a suboptimal level.^{11,12} Rana et al emphasized that pain management is still not an essential component of oncological care.¹³ The addition of a fourth step consisting of interventional procedures to the WHO's three-step analgesic ladder has been suggested.^{14,15} It should be recognized that intractable pain is a humanitarian as well as a medical problem, and especially in cancer patients, this problem can be solved with rational treatment alternatives. Pain-free survival has been defined as a human right by both the WHO and the International Association for the Study of Pain (IASP)¹⁶; however, it is a sad fact that this right is not recognized adequately in the present day. Human beings should be given the right to die with the same dignity with which they are accorded by law to live.

The first attempt at destructive procedures for intractable pain treatment was originally performed in 1912 using open surgical techniques in the spinal cord.² In 1963 Sean Mullan described the percutaneous approach for performing cordotomy. Beginning in 1965, RF energy was used to make controlled lesions.^{17,18} Later, the RF generator and electrode systems provided information regarding use of the impedance of the tissue and electrical stimulation to evaluate the function of the target.¹⁹ These procedures, called "stereotactic," were classically performed using radiographic guidance; however, it was determined later that it was impossible to visualize the spinal cord and the target using such conventional visualization methods.²⁰ Finally, in response to the necessity of a dynamic visualization system, we started to use CT guidance in percutaneous procedures.²¹⁻²³

The descending trigeminal tract is an excellent target for controlling intractable facial and oronasopharyngeal pain. Destruction of the descending trigeminal tract in the medulla is known as a trigeminal tractotomy.¹ Lesioning of the nucleus caudalis is known as trigeminal nucleotomy, and lesioning of the whole substantia gelatinosa of the nucleus caudalis is known as the nucleus caudalis dorsal root entry zone (DREZ) operation.²⁴⁻²⁶ The pain-transmitting fibers of cranial nerves VII, IX, and X and descend with the spinal tract of the trigeminal nerve into the upper spinal cord.^{27,28} Lesioning of this tract was first performed by Sjöqvist in 1938.¹ In 1965 Kunc successfully used the procedure to relieve glossopharyngeal neuralgia.²⁹ Sweet observed special hypoalgesia in the dermatomes of cranial nerves VII, IX, and X after trigeminal tractotomy.³⁰ Crue et al and Hitchcock, with the aid of RF thermocoagulation, independently performed the first stereotactic trigeminal tractotomies.^{31,32} Schvarcz, who has used the technique since 1971, suggested lesioning the oral pole of the nucleus caudalis, and designated the procedure as trigeminal nucleotomy.^{24,33,34} Hosobuchi and Rutkin used evoked potentials to delineate the descending trigeminal tract during the procedure.³⁵ In 1990 Nashold's group³⁶ described a special open technique to destroy the substantia gelatinosa of

the nucleus caudalis, and named the procedure the trigeminal nucleus caudalis DREZ operation.^{25,26} In 1989 we developed a percutaneous technique for trigeminal tractotomy-nucleotomy (TR-NC), guided by CT, to facilitate topographical localization of the electrode tip in the spinal cord.²³

In this chapter, our vast experience with trigeminal TR-NC with CT guidance is presented in conjunction with the knowledge gained from our patients and colleagues.

■ Pertinent Anatomy and Rationale

The descending trigeminal tract is the caudalward branch of the trigeminal afferents at the medulla after its bifurcation upon entry into the pons. These fibers terminate in the spinal trigeminal nucleus.^{1,37} The tract overlies the spinal trigeminal nucleus in the posterolateral part of the spinal cord at the cervicomedullary junction. All three divisions of the trigeminal nerve have a specific topographic organization in the tract: fibers from the mandibular dermatome (V-3) are located in the dorsal part of the tract; ophthalmic fibers (V-1) are located ventrolaterally, and maxillary fibers (V-2) are located between them. Primary sensory fibers from cranial nerves VII, IX, and X also enter the tract.^{38,39} These fibers lie slightly medially, behind the tract. The trigeminal sensory nucleus consists of three nuclei: (1) spinal, (2) principal, and (3) mesencephalic trigeminal nuclei. Afferent fibers that carry the sensations of pain and temperature descend in the spinal trigeminal tract in the medulla and terminate in the spinal trigeminal nucleus.

The spinal trigeminal nucleus has three distinct subdivisions along its pontospinal extent: (1) the nucleus oralis, located rostrally between the pons and medulla; (2) the nucleus interpolaris, located intermedially; and (3) the nucleus caudalis, located at the medullospinal junction and extending down to the level of the C-2 segment.⁴⁰ The nucleus caudalis represents the substantia gelatinosa, and there is an extensive overlap between facial and high cervical afferents, where the 7th, 9th, and 10th afferents also end. From a neuropathological basis, the secondary caudalis neurons begin to fire like those in an epileptogenic area after deafferentation, as in anesthesia dolorosa, postherpetic pain, and trigeminal dysesthesia.⁴⁰ Schwarcz attributed particular importance to the destruction of the oral pole of the nucleus caudalis, which probably acts on the pathology site, removes the pool of neuronal hyperexcitability, eliminates convergence, and severs the ascending intranuclear polysynaptic pathways.^{24,34} The nucleus caudalis of the trigeminal nerve lies between a point 5 mm above the obex at the posterolateral part of the bulbus and spinal cord and the dorsal root of the C-2 segment (approximately 20 mm below the obex). At the medullobulbar junction, it is located between the lateral border of the dorsal column (fasciculus cuneatus) and the rootlets of the spinal cord in the axial section.^{26–28,37}

There is a topographic representation of the ipsilateral face on the spinal tract of the trigeminal nerve; that is, the most central areas of the face terminate highest on the nucleus caudalis and the most peripheral areas of the face end lowest. Called onion-skin organization, this causes the central area of the face to be spared from hypoalgesia after the nucleus caudalis DREZ operation if the lesions do not extend above the obex.^{25,27,28,37,40}

The dorsal spinocerebellar pathway is located immediately lateral to the descending trigeminal tract and nucleus caudalis, and the external arcuate fibers cover the tract posteriorly; thus, ataxia of the ipsilateral extremities usually accompanies extensive tractotomy and nucleotomy.^{25,26,38,40} The lateral spinothalamic tract is located anteriorly to the descending trigeminal tract and the nucleus caudalis. Anterior lesions may produce analgesia on the contralateral body. The funiculus cuneatus is located just posteromedially to the descending cranial nociceptive tract and the 7th, 9th, and 10th fibers, and a lesion involving the funiculus may produce loss of proprioceptive sensation in the lower extremities.^{40,41}

■ Indications

Destructive procedures on the descending cranial nociceptive tract and the trigeminal nucleus caudalis are indicated in patients with craniofacial paroxysmal, dysesthetic, or deafferentation pain, especially in anesthesia dolorosa; postherpetic dysesthesia; atypical facial pain; dysesthetic sequelae after previous trigeminal surgery; posttraumatic neuropathy; geniculate-glossopharyngeal neuralgias; and head, neck, or facial pain from malignancy.^{1,28,29,32–34,41–45} Patients with head, neck, or facial pain due to malignancy may be the best candidates for such operations.

■ Technique

Patients should be fasted for 5 hours before the operation. If required, neuroleptic anesthesia should be given at a dose that will not affect patient cooperation during the procedure.⁴⁰ In each case, a cranial CT scan must be taken to rule out a mass lesion due to metastasis because this would be a contraindication to performing TR-NC. It is important to stress that all patients should be well informed regarding the possibility of a midprocedure cancellation if they are unable to tolerate the operation. In our series, such cancellations were necessary in four patients (4.9%).

Target

The target is the descending trigeminal tract and the nucleus caudalis, located at the medullospinal junction at the occiput–C1 level (**Fig. 19.1**). The descending trigeminal tract and nucleus caudalis are located laterally and anteriorly to the fasciculus cuneatus in the posterolateral part of the upper spinal cord, and the posterior spinocerebellar tract lies just outside the descending trigeminal tract.⁴⁰ The nucleus caudalis is located medially to the descending trigeminal tract, and the trigeminal tract is located laterally. Within the tract, the facial, glossopharyngeal, and vagal fibers are located posterolaterally, whereas the trigeminal fibers are located anterolaterally. The target is 3 mm deep from the posterior aspect of the spinal cord and in the lateral third of the transverse diameter of the semicord. Because of the close proximity between the nucleus caudalis and descending trigeminal tract, any lesion usually affects both structures; thus, we use the term TR-NC.⁴⁰

Needle and Electrode System

The Kanpolat cannula and electrode system are recommended for this procedure (Cosman, Burlington, MA, USA). Straight and curved electrodes with a 0.4-mm diameter and 2-mm open tip are usually used; rarely, 0.3-mm-diameter, 2-mm open-tip straight and curved electrodes are chosen.^{40,46}

Injection of Contrast Material

Generally, contrast material is administered into the subarachnoid space of the spinal cord by lumbar puncture 20 to 30 minutes before the procedure. A lateral scanogram is obtained, axial CT scans using a 1-mm slice thickness are taken, and the spinal cord diameters at the occiput–C1 level are measured.⁴⁰

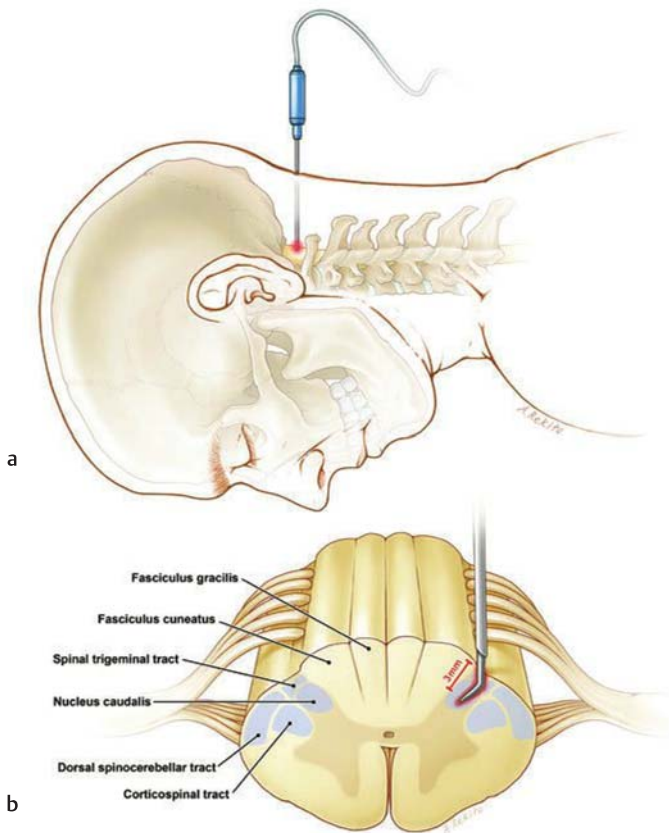


Fig. 19.1 Schematic representation of the trigeminal tractotomy-nucleotomy (TR-NC) procedure. **(a)** Insertion of the needle into the occiput–C1 level in prone position. **(b)** Final position of the electrode tip in the target and pertinent anatomical structures.

Positioning

The patient is placed on the CT table in the prone position. With the help of the head support of the CT table, the patient's head is kept in slight flexion. The chest must be elevated and supported with soft pads, the head is fixed with a fixation band, and nasal oxygen tubing is fixed to the patient's nostrils.⁴⁰

Insertion of the Needle Electrode System

The needle is inserted at the occiput–C1 level, 7 to 8 mm lateral to the midline. Local anesthetic is administered before the initial puncture. Special attention must be given to ensuring the cannula is kept straight during the puncture. Placement of the needle at the occiput–C1 level can be seen in the lateral scanogram (**Fig. 19.2a**). The distance between the dura and the skin has been measured with CT scans and ranges from 40.5 to 56.5 mm (mean, 49 mm).^{40,47} This critical range must be kept in mind during the procedure. The dura is punctured, and the cerebrospinal fluid (CSF) gradually emerges. The needle must be positioned in the posterior aspect of the spinal cord, one-third lateral to the semicord (**Fig. 19.2b**). The adjusted active electrode tip is inserted into the spinal cord using the needle (**Fig. 19.2c**). The inserted part of the active electrode is adjusted in accordance with the spinal cord diameters measured at the beginning of the procedure. The puncture should be done as gently as possible; even so, the patient must be warned that the puncture will be painful. Impedance measurements are taken and should be less than 400 ohms when the electrode system is in the CSF, and greater than 1,000 ohms when the electrode system is inside the spinal cord.⁴⁰

Stimulation

Electrical stimulation with low (2–5 Hz, 0.1–0.3 V) and high (50–100 Hz, 0.2–1.0 V) frequencies is used. We recommend starting the stimulations in low frequencies. Observation of paresthesia of the face is a good indication that the electrode is located in the trigeminal fibers. If the 5th, 9th, and 10th fibers are the targets, slight withdrawal of the tip and restimulation usually cause a dysesthetic sensation in the throat or inside the ear, indicating that the tip is in the nociceptive fibers of these cranial nerves (IX and X).⁴⁰

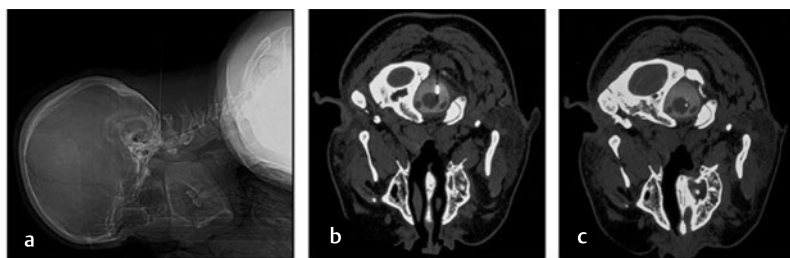


Fig. 19.2 The CT-guided trigeminal tractotomy-nucleotomy (TR-NC) procedure. (a) Position of the needle at the occiput–C1 level in the lateral scanogram. (b) Final position of the needle in the axial CT scan. (c) Final position of the electrode.

Lesioning

It must be kept in mind that TR-NC lesioning is painful. At this stage, if the surgeon is certain, according to the CT slices, that the electrode is in the correct position, and stimulation confirms these findings, neuroleptic anesthesia is administered. The temperature of the electrode is increased gradually. The first lesion is made at a temperature of 50 to 60°C to 80°C for 60 seconds. One or two additional lesions are made if necessary.⁴⁰

■ Outcomes

Eighty-one patients who underwent 96 CT-guided TR-NC procedures were followed for 1 to 216 months (mean, 64.8 months). Patients were followed via telephone calls or direct visits, and the period was ended in the event of any change in their contact information or their death. The distribution of their primary diseases is shown in **Table 19.1**. The term *mixed craniofacial pain* was attributed to the situation of trigeminal neuralgia, which evolved later into glossopharyngeal neuralgia in one case and into geniculate neuralgia in another. Complete pain relief indicated maintenance of a pain-free status in the patient without the use of any medication. Partial pain relief indicated positive response to the procedure, with the pain controlled with a lower medication dose than

Table 19.1 Distribution of patients according to their primary pathologies and the status after the TR-NC procedure in the early follow-up period

Pathology	n (%)	Not tolerated n	Complete pain relief, n (%)	Partial pain relief, n (%)	Failed pain relief, n (%)
Atypical facial pain	16 (19.7%)	1	8 (53.3%)	3 (20%)	4 (26.7%)
Atypical trigeminal neuralgia	12 (14.8%)	–	11 (91.7%)	1 (8.3%)	0 (0%)
Geniculate neuralgia	6 (7.4%)	–	5 (83.3%)	0 (0%)	1 (16.7%)
Glossopharyngeal neuralgia	20 (24.7%)	–	17 (85%)	1 (5%)	2 (10%)
Malignancy	17 (21.0%)	3	10 (71.4%)	2 (14.3%)	2 (14.3%)
Bilateral trigeminal neuralgia	2 (2.5%)	–	2 (100%)	0 (0%)	0 (0%)
Anesthesia dolorosa	2 (2.5%)	–	0 (0%)	1 (50%)	1 (50%)
Postherpetic neuralgia	4 (4.9%)	–	2 (50%)	1 (25%)	1 (25%)
Mixed craniofacial pain	2 (2.5%)	–	2 (100%)	0 (0%)	0 (0%)
Total patients	81 (100%)	4 (4.9%)	66 (81.5%)		11 (13.6%)

Abbreviation: TR-NC, trigeminal tractotomy-nucleotomy.

previously necessary. The last group consisted of those cases that responded to the procedure poorly or not at all.

In 4 patients (4.9%), the procedure was terminated due to patient intolerance. Complete or partial satisfactory pain control, which was accepted as success, was obtained in 66 patients (85.7%). Patients who responded to the procedure poorly or not at all were classified as failure (14.3%). The majority of the patients had glossopharyngeal neuralgia, with a success rate of 90%. The results of the TR-NC procedure immediately afterward and in the early follow-up period (up to 1 month after the procedure) are shown in **Table 19.1**. The best results were obtained in patients with geniculate neuralgia; complete pain relief was achieved in 5 of 6 patients, and the remaining patient required the NC-DREZ operation additionally to obtain the same result. The worst results were determined in patients with anesthesia dolorosa.

At the conclusion of the follow-up period, complete pain relief was obtained in 55 patients (71.4%), partial pain control in 13 (16.9%), and failure in 9 (11.7%).

In the total series, additional procedures were required after the TR-NC due to intractable pain in 14 patients during the follow-up period (DREZ lesioning operation, $n = 10$; neurotomy, $n = 2$; microvascular decompression, $n = 1$; RF rhizotomy, $n = 1$). Complete pain relief was obtained in all patients who had additional procedures except in the DREZ group. One patient who suffered from intractable facial pain due to invasive pituitary tumor did not respond to either the TR-NC or the DREZ operation and later completed suicide. Of the remaining 9 patients who underwent DREZ, partial pain relief was obtained in 2 and complete pain relief in the remaining 7. One patient who suffered from intractable pain due to geniculate neuralgia responded partially to the TR-NC procedure and underwent the NC-DREZ operation. He was pain free following the DREZ operation, but was lost due to pulmonary embolism prior to discharge.

Complications

CT-guided TR-NC is an effective and safe procedure, with a low risk of complications. In tractotomy and nucleus caudalis DREZ lesioning, the most important complication is ataxia, caused by lesioning of the dorsal spinocerebellar tract.^{25,26,48} Contralateral hypoalgesia is observed as a complication in cases of lesioning of the lateral spinothalamic tract if the lesion is made anterior to the target.⁴⁸ In our series, temporary ataxia was the only complication; it was observed in 4 cases (4.2%), and all cases resolved within 2 weeks.⁴⁰ Within the series of the author spanning the past 20 years, the procedure-related mortality rate is zero.

Conclusion

Descending trigeminal tractotomy, the nucleus caudalis DREZ operation, and trigeminal nucleotomy are different operative techniques for destruction of the descending trigeminal tract, the substantia gelatinosa of the nucleus caudalis, and a special, limited part of the nucleus caudalis.^{26,31,32,34,38} These procedures may involve open surgical techniques, that is, craniectomy or upper cervical laminectomy and dural opening, or percutaneous lesioning with a needle-and-electrode RF system. We prefer to call the technique TR-NC because there is still a lack of

anatomical and physiological data as to which anatomical part of the involved structures, the nucleus or the tract, is destroyed and which part is the cause of pain relief.

Using CT guidance allows direct visualization of the upper cervical spinal cord at the occiput–C1 level. Theoretically, the nucleus caudalis should be destroyed in patients with neuropathic pain, and the descending trigeminal tract should be destroyed in patients with neuralgic pain. Therefore, the aim is slightly lateral to the nucleus–tract complex when the intention is to destroy the tract and slightly medial for destruction of the nucleus.

For medically intractable cases in which typical trigeminal neuralgia is present but has not been managed with customary surgical techniques, CT-guided TR-NC is not the operation of choice.⁴² Cases classified as failed trigeminal neuralgia, which present great problems in their management, may be treated with this procedure. Most of these patients have been managed previously with operations such as microvascular decompression, RF rhizotomy, and intragasserian glycerol injection, and they usually have neuropathic pain or even anesthesia dolorosa. CT-guided TR-NC should be performed in these patients before the trigeminal nucleus caudalis DREZ operation, which is a much riskier and more invasive procedure.^{25,26,48,49}

In my practice, distinguishing facial pain as type II or atypical facial pain depends on my years of experience. In other words, this is still a controversial issue.⁵⁰ Clarification is possible with the use of a multidisciplinary approach between the neurosurgeons and neurophysiologists. Comparable with the chronicity of a craniofacial disease, it is notable that the pain character may change in time, from neuralgic to neuropathic or vice versa.

I believe that trigeminal neuralgia is a lifelong disease. As with its main cause, the causes of its recurrence are unknown. The incidence of bilateral cases was 2.95% in our series.⁵¹ Patients with bilateral trigeminal neuralgia who are older (6th decade or more) or who have a medical problem that presents a relative contraindication to a more invasive surgical procedure with general anesthesia, may be candidates for CT-guided TR-NC. Usually, these patients have undergone RF rhizotomy on one side and may have moderate or severe hypoesthesia on that side.⁴ A contralateral RF rhizotomy may cause severe difficulties in mastication, and CT-guided TR-NC, which preserves sensory modalities other than pain, may be performed on that side.

Microvascular decompression^{52,53} is effective for treating glossopharyngeal, vagal, or geniculate neuralgias, but its mortality and complication rates are worth regarding as important risk factors. In our series, CT-guided TR-NC was effective in relieving pain in most cases with glossopharyngeal and geniculate neuralgia.⁵⁴ Combined neuralgic involvement of cranial nerves VI, VII, IX, or X presents a serious problem as to surgical treatment, and we propose that CT-guided TR-NC may be the most suitable choice.

Patients with craniofacial malignancies may have severe, intractable cranial neuralgic or neuropathic pain, and may be especially difficult to manage. CT-guided TR-NC is the ideal procedure in such cases.⁵⁵ Tissue changes attributable to previous surgical procedures or applied radiation may present some difficulties for CT-guided TR-NC.

CT-guided TR-NC is also effective in the treatment of patients with atypical facial pain. Such patients should be observed carefully before any kind of surgical intervention is done.⁶

The indications for CT-guided TR-NC are similar to those for the nucleus caudalis DREZ operation, but the effectiveness of the former procedure is quite high and the complication rate is extremely low.²⁵ TR-NC is safe and effective for repeated applications. The DREZ operation may be performed only if TR-NC is not sufficiently effective. With its advantages of minimal invasiveness, high success rates, and low complication rates, percutaneous CT-guided TR-NC should be considered as a rational alternative to other pain-relieving procedures.

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Video 3. Percutaneous CT-Guided Trigeminal Tractotomy and Nucleotomy

Editor's Comments

Professor Kanpolat has made major contributions to the field of pain surgery, not the least of which is his creative and staunch support of percutaneous ablative procedures. His innovations have been solidly based in anatomy, and this chapter highlights that approach. He has shown us that a superior understanding of neuroanatomy and readily available technology can combine for high-quality outcomes, opposing the most difficult pain syndromes and enhancing patient comfort. No one in the world deserves more credit for maintaining the flame of knowledge and experience in this area.

The TR-NC (trigeminal tractotomy-nucleotomy) procedure appears, in his hands, to be a highly effective option for patients with medically intractable craniofacial pain. He includes in the indications for the procedure "paroxysmal, dysesthetic, or deafferentation pain, especially in anesthesia dolorosa; postherpetic dysesthesia; atypical facial pain; dysesthetic sequelae after previous trigeminal surgery; posttraumatic neuropathy; geniculate-glossopharyngeal neuralgias; and head, neck, or facial pain from malignancy." Each of these conditions poses unique challenges. If TR-NC can be successfully passed on to the next generation of pain surgeons and helps even 50% of these patients, it would represent a major breakthrough.

My hope is that through this chapter, and accessible video documentation linked to it, other pain surgery practitioners will come to adopt this technique, and determine if it is equally successful in their hands and for their patient populations.

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Section II.E

Destructive Procedures

20

Overview of Destructive Neurosurgical Procedures for Pain

Daniel R. Cleary and Justin S. Cetas

Targeted lesions made in the nervous system can provide pain relief when medical management and neuromodulation are no longer sufficient. Destructive techniques are primarily used to treat medically intractable cancer pain, where life expectancy is limited, but in some cases they are also useful for long-term relief from nonmalignant pain. Before proceeding with any ablative procedure, medical and neuromodulatory options should be thoroughly explored. Destructive procedures should be used selectively and with careful consideration of patient circumstance because neuroablation has the potential to worsen pain or cause permanent disability. Ablative techniques are best suited to cases where neurological deficiencies are already present, where new deficits will have less of an impact on quality of life, or where the potential benefit strongly outweighs the potential adverse effects. With late-stage malignancy, destructive procedures are used because a shortened life expectancy decreases the potential harm from a surgical complication or adverse effect. In contrast, with nonmalignant pain, destructive techniques should be considered only when drastic improvements in quality of life and few adverse effects are expected.

Destructive neurosurgical techniques produce analgesia by destroying overactive areas of the nervous system or by blocking nociceptive signaling pathways. Attempts have been made with varying success to disrupt nociceptive signaling at nearly all levels of the nervous system, from peripheral nerves to selected regions of cerebral cortex (**Fig. 20.1**). Few rigorous clinical studies have been done on destructive procedures for pain management, and reported short-term success rates range

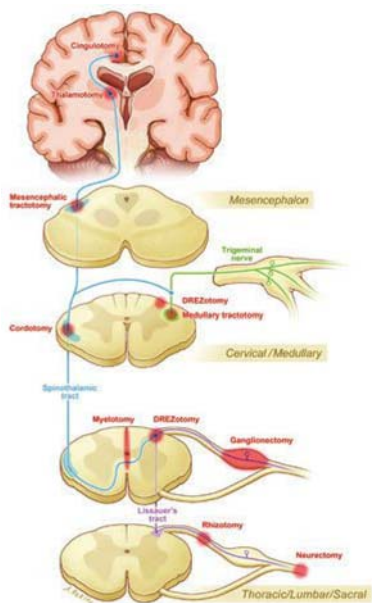


Fig. 20.1 Common ablative sites for the treatment of intractable pain. Lesions can be made along all levels of ascending nociceptive transmission, from peripheral neurectomy to cingulotomy, with varying degrees of success.

from as low as 20% of patients receiving pain relief to greater than 90% patient satisfaction. Outcomes vary depending on patient selection, the indications for the procedure, and the experience of the surgeon with the technique, but the percentage of patients who continue to experience relief consistently declines in the months to years after the procedure. Patient evaluation is a critical factor for a successful surgery, and the procedure should be well matched to the mechanism, quality, and distribution of the pain. Somatic or cancer pain that responds to opioid therapy is likely to be helped by ablation of sites in the canonical nociceptive pathways, as with cordotomy or rhizotomy, whereas little improvement in neuropathic pain is seen with lesion to these same sites. Similarly, lesion to the dorsal root entry zone is highly effective for deafferentation pain from brachial plexus avulsion, but performs poorly for postherpetic neuralgia.

■ Techniques and Outcomes

To combat chronic pain, neurosurgical procedures have been developed to target nearly every level of the nervous system, from peripheral nerves to frontal cortex. This overview of techniques and outcomes follows the transmission of nociceptive signals from peripheral to central, with discussion emphasis on procedures more commonly used.

Destruction of First-Order Neurons: Neurectomy, Rhizotomy, and Ganglionectomy

Destruction of sensory nerves and ganglia produces immediate anesthesia and pain relief, but patients often have poor long-term outcomes and worsening of pain due to the regrowth of nerves or the development of alternative afferent pathways.¹⁻⁴ Destruction of first-order neurons also often results in such adverse effects as dysesthesias, loss of proprioception, and occasional loss of motor function,⁵ so these procedures should be selectively applied.

Neurectomy, the destruction and removal of a peripheral nerve, provides reasonable short-term pain relief with variable long-term results.^{6,7} Common long-term side effects include the development of deafferentation syndromes or neuromas; boundary pain; recurrence of pain; and loss of motor, sensory, or proprioceptive function. Because of these adverse effects, this procedure is not usually performed for pain in the extremities, but instead is primarily used for facial pain, trigeminal neuralgia, and some post-surgical pain syndromes.⁸⁻¹¹

Rhizotomy, the partial or complete destruction of the dorsal nerve root, has similar outcomes as those that occur with neurectomy and ganglionectomy. Complete rhizotomy is rarely used due to frequent adverse effects and poor pain control, but partial rhizotomy, specifically lesion of the medial branch of the dorsal root, is used for a variety of pain syndromes. Medial branch lesion is not recommended for extremity pain, but has been successfully used for trigeminal neuralgia, lumbar facet syndrome, cervical pain, cluster headache, and malignant pain.¹²⁻¹⁴ Sufficient evidence exists to recommend medial branch ablation for select patients because greater than half of patients usually receive therapeutic benefit.^{6,15,16} As with other destructive procedures, the high success rates seen early after the procedure may decline over time.¹⁰

Ganglionectomy, the removal of the entire dorsal root ganglion, has been advocated as an improvement upon rhizotomy with more complete pain control. This idea is based on observations of sensory and unmyelinated afferents entering the spinal cord via the ventral root, which could explain why some patients obtain only minimal pain relief after complete rhizotomy.^{17,18} Ganglionectomy has been tried for many of the same indications as rhizotomy, including both malignant and nonmalignant pain syndromes. In one of the few randomized, double-blinded trials for surgical pain control, ganglionectomy produced no better pain control than placebo for nonmalignant back pain, so the procedure is strongly discouraged for treatment of lumbosacral radicular pain.^{5,19} Other outcome reports for ganglionectomy are mixed, and it is unclear whether the procedure offers clear improvement over partial rhizotomy.^{6,16,20} Overall, ganglionectomy is a relatively safe procedure with low morbidity and fair pain relief, but few patients have prolonged pain relief.^{10,21}

Lesion of Dorsal Root Entry Zone (DREZotomy)

Among destructive neurosurgical procedures for pain control, lesion of the dorsal root entry zone (DREZ) is one of the most recently developed and most effective. The basic technique was first described by Sindou²² and later modified by Nashold to include a greater area of ablation.²³ The modern DREZotomy procedure destroys the central portion of the dorsal rootlets, the most superficial layers of the dorsal horn, and the superficial band of fibers transmitting nociceptive inputs (Lissauer tract). It is typically done as an open procedure using a hemilaminectomy, and multiple lesions are made in a rostral-caudal distribution.

In selecting patients for DREZ ablation, their pain should be unilateral and with a limited distribution because this technique does not provide pain relief above or below the region affected by the lesion. DREZotomy is highly efficacious in the treatment of deafferentation pain from brachial plexus avulsion, spinal cord injury, or tumor invasion, and has also successfully been used with malignant pain and pain from hyperspasticity, albeit with less success.^{13,24} The procedure has poor to moderate outcomes for treatment of phantom limb pain, stump pain, and postherpetic neuralgia. In cases of deafferentation pain with brachial plexus injury, DREZ ablation should be considered as a first-line treatment because more conservative management is less frequently successful. Case studies exist with reports of greater than 90% of patients having excellent relief at discharge, and the majority of patients continue to have acceptable pain relief several years after surgery.^{6,25-27} Side effects of DREZotomy are infrequent and often transient, but can include permanent motor deficits, dysesthesias, and loss of bowel or bladder function.²⁸

Myelotomy

The commissural myelotomy is a destructive technique that blocks signal transmission at the spinal decussation of the ascending nociceptive afferents. Second-order nociceptive neurons from the dorsal horn extend axons ipsilaterally for one to two spinal levels, and then these fibers cross the midline at the median commissure. Selective disruption of these fibers provides bilateral pain relief covering a broader area than DREZ lesion, although the coverage is not as extensive as with cordotomy. The commissural myelotomy is used less frequently

than other destructive methods because the requisite laminectomy is not well tolerated by sick patients, and neurologic complications are common.²⁹ The most common adverse effects are loss of bowel and bladder function, and loss of motor, sensory, and proprioceptive function. Recurrence of pain frequently occurs in the months after surgery, and so commissural myelotomy is not recommended for nonmalignant pain. Clinical studies on commissural myelotomy are limited to case reports, but usually greater than half the patients report adequate pain relief from the procedure.^{6,16}

The limited midline myelotomy and the punctate midline myelotomy were modifications of the commissural myelotomy made in an effort to improve outcomes and reduce complications.^{30–33} These techniques are particularly effective at relieving midline or visceral pain, possibly by targeting a previously unrecognized ascending visceral nociceptive pathway deep in the midline of the dorsal columns.^{34,35} The midline myelotomy and modifications provide pain control for bilateral or visceral pain syndromes, such as with abdominal malignancy, but the technique does not often improve somatic pain and so is limited in the scope of application. Only a relatively small laminectomy and spinal lesion are required, which are generally well tolerated by most patients. Fair to good pain control is often achieved with few adverse effects, but evidence is largely limited to case reports.^{36–38}

Cordotomy

Unilateral lesion to the ascending nociceptive afferents in the spinothalamic tract (STT) provides more effective and widespread pain relief than myelotomy, although complications can also be more severe. Cordotomy targets the STT at the cervical level, thereby providing effective pain relief for thoracic, lumbar, and lower cervical regions without compromising sensory function. It is primarily indicated for pain limited to a unilateral distribution, but a bilateral procedure can be used for midline, visceral, or otherwise broadly distributed pain. Cordotomy is most often performed for pain from malignancy or spinal cord injury and is most effective in cases where the pain has a clear nociceptive origin. Although it is occasionally performed for nonmalignant pain, cordotomy often does not provide adequate relief for persistent neuropathic pain and can have life-threatening complications.³⁹ The most significant risk of cordotomy is central apnea (Ondine curse) due to the proximity of the STT to descending fibers controlling respiration. This complication occurs more frequently and tends to be more severe following bilateral ablation. Nearly all patients who receive cordotomy will temporarily have Horner syndrome, and a lower number will have unilateral motor dysfunction, loss of bowel and bladder control, dysesthesias, and, more rarely, unmasking of contralateral pain. Most of these problems are transient, but the potential remains for permanent dysfunction.

Percutaneous cordotomy is now one of the most commonly used destructive techniques for intractable pain, and it has some of the highest levels of patient satisfaction and longest durations of effect—as long as 35 pain-free years in one case.^{16,40,41} The percutaneous technique is less invasive than prior methods and requires only the introduction of an electrode into the cervical spinal cord at the level of C1–C2. The STT can be accurately targeted using myelography with fluoroscopy, impedance measurements, or intraoperative stimulation. The recent

introduction of intraoperative computed tomography (CT) guidance for electrode localization has led to significant improvements in targeting and thus fewer complications.^{38,41,42}

Outcomes of cordotomy stand out among other destructive procedures for pain control, and patient satisfaction with the use of CT guidance is reported to be as high as 98%, with significant improvements in pain, in activities of daily living, and with sleep.^{6,40} No randomized, controlled trials have been done on cordotomy, but case studies frequently show complete pain resolution in a high percentage of patients.^{42–44} With a carefully selected cohort, cordotomy is a highly efficacious technique for the treatment of intractable pain.

Medullary Lesions

Destructive procedures to treat chronic facial pain target nociceptive-specific structures within the medulla, including the nucleus caudalis and the descending trigeminal tract. The nucleus caudalis is primarily involved in processing nociceptive input from the trigeminal (gasserian) ganglia, but it also receives some sensory and nociceptive input from cranial nerves VII, IX, and X. The descending trigeminal tract is a group of afferent fibers from the gasserian ganglion that enter at the pons and transverse caudally to synapse within the nucleus caudalis.

Two similar techniques targeting these regions are commonly used with moderate to good results. Stereotaxic trigeminal tractotomy/nucleotomy targets the descending trigeminal tract and/or the nucleus caudalis, and is done as a percutaneous procedure using CT guidance or microendoscopy.^{45–47} Nucleus caudalis DREZ ablation is a similar procedure that targets the second-order nociceptive neurons of the nucleus caudalis, although the caudalis DREZ ablation is an open microsurgical procedure and uses numerous small lesions to destroy the entire nucleus caudalis.^{48,49} Both operations have been used for cancer pain, postherpetic neuralgia, intractable trigeminal neuralgia, anesthesia dolorosa, and atypical facial pain, all with varying degrees of success. The most frequent complication is ataxia due to damage to the nearby spinocerebellar tract.

A clear indication does not exist for one procedure over the other, although the less invasive percutaneous technique may be better tolerated by patients with late-stage malignancy. Because the nucleus caudalis is primarily involved in nociceptive processing, patients typically have analgesia without anesthesia. As with other ablative procedures, pain control often lessens in the months to years after the procedure.^{10,50} With careful selection, most patients see some improvement in pain control, although evidence is limited mostly to case reports and a small number of controlled studies. With some forms of atypical and neuropathic facial pain, earlier intervention correlates with greater benefit, and so with nerve injury these procedures should be considered before extensive deafferentation occurs.

Mesencephalic Tractotomy

Similar in concept to cordotomy, lesions to ascending STT fiber tracts within the mesencephalon have been used to treat intractable pain. Mesencephalic tractotomy has been tried for nociceptive, deafferentation, and central pain syndromes, including poststroke pain, thalamic syndrome, facial pain, and cancer pain.⁵¹ Although studies are limited, the procedure has high reported success

rates for treating nociceptive and malignant pain (upwards of 85%) but poor outcomes with neuropathic and deafferentation pain.^{6,51,52} The ablative target is in close proximity to the medial lemniscus, so sensory loss and dysesthesia are common side effects, and ocular palsies and diplopia can also occur. This technique has some advantages over cordotomy because it offers pain control at the facial and high cervical levels, has a lower risk of motor and respiratory side effects, and can be more safely performed bilaterally. Although with recent improvements in outcomes with cordotomy, mesencephalotomy now is rarely indicated over cordotomy.

Thalamotomy

Ascending nociceptive fibers terminate in the medial and lateral thalamus, which respectively project to areas involved in the affective and somatosensory aspects of pain.^{54–56} Lesions of both the medial and lateral thalamus have been tried for multiple forms of pain, but only lesions of the medial thalamic nuclei reliably provide pain relief.²¹ The therapeutic mechanism of thalamotomy is unclear, but the projections of the medial thalamus to the prefrontal and anterior cingulate cortices may be involved in therapeutic effect. Medial thalamotomy is effective for the treatment of malignant, central, neurogenic, and deafferentation pain, although outcomes vary and reemergence of pain frequently occurs over time. Evidence in favor of this technique for pain control is largely limited to case series, but reports consistently show a majority of patients receive pain relief for a few months to a few years after surgery.

Thalamic lesions are typically made using a stereotaxic approach and radiofrequency coagulation, but they can also be successfully performed noninvasively using Gamma Knife (Elekta, Stockholm, Sweden) or high-intensity focused ultrasound.^{57–59} Regardless of the method used, medial thalamotomy is among the safest of the destructive neurosurgical procedures for pain.

Cingulotomy

Stereotaxic ablation of the cingulate cortex does not produce analgesia or decreased sensory function, but instead appears to decrease the affective component of pain. The cingulate cortex is involved in the perception and affective processing of pain, and the region has connections to the medial thalamus, the midbrain periaqueductal gray, and other areas of the cortex.^{60,61}

The beneficial effects of cingulotomy on pain were first noted in patients with “unbearable pain” who subsequently received prefrontal lobotomy.⁶² Since that time, cingulotomy has been performed for many pain-related conditions, and the procedure has uses in the treatment of central pain, malignant pain, and some forms of neuropathic pain. About half of patients receiving the procedure will see lasting therapeutic improvement, including decreases in reported visual analog pain scale ratings, decreased use of and craving for opioids, and pain becoming less bothersome.^{6,16,63} These benefits tend to fade in the years following the procedure, so repeat cingulotomies or additional synergistic surgical procedures can be performed to prolong and improve the therapeutic effect.^{64–66}

Despite the destructive nature of cingulotomy, adverse neurological effects are infrequent and rarely permanent. Immediately after surgery, patients most commonly experience headache, bladder dysfunction, seizures, and postsurgical confusion. Long-term changes in attention, learning, organization, and motivation have also been observed, although these latter changes are often subtle.^{63,67} Because this procedure involves irreversible destruction of an area of the forebrain, it should be considered only when conventional treatments have failed.

Sympathectomy

In complex regional pain syndrome (CRPS), abnormal sympathetic activity causes burning pain (causalgia), hyperalgesia, and abnormal vascular function. These symptoms are poorly responsive to opioid analgesics but can be effectively relieved by medical or surgical sympathetic blockade.^{68,69} Interruption of sympathetic activity by division of the paravertebral sympathetic ganglion chain can permanently relieve chronic pain, especially when treatment is initiated early in the disease process.^{70,71} Sympathectomy is one of the few destructive neurosurgical techniques for nonmalignant pain that is indicated early in the disease process and consistently offers a high degree of success. Studies on sympathectomy include well-controlled trials and case reports, and strong evidence shows sustained improvements in pain for a high percentage of patients with CRPS.⁷² In light of the high rate of definitive improvement following sympathectomy for CRPS, misdiagnosis should be considered in cases of poor outcome following surgery. The procedure also has some utility in the treatment of malignant pain from pancreatic and upper abdominal visceral malignancies, but tends to be poorly efficacious for the treatment of neuropathic pain and nonsympathetically mediated pain syndromes. Common complications include hyperhidrosis, Horner syndrome, vascular injury, and the reemergence of pain over time.⁷³

Conclusion

Destructive neurosurgical procedures for pain control target nociceptive neural pathways and dysfunctional elements of the nervous system. When medical therapies are no longer sufficient, neurological lesions offer long-lasting pain relief, although the potential for permanent neurological dysfunction is often high. In-depth discussion with the patient should be made prior to any destructive neurosurgery, and care should be taken to ensure the surgery is appropriately matched to the pain syndrome. Patient outcomes are often mixed, with some patients seeing no improvement, whereas others may have a dramatic decrease in pain. For nearly all destructive procedures for pain, the therapeutic effect will decrease over time. Improved outcomes and lower complication rates have been seen in recent years through refinements of technique and an increased use of intraoperative imaging, but a strong need still exists for randomized and well-controlled clinical trials.

Editor's Comments

The history of pain surgery virtually begins with procedures that intentionally damaged “pain pathways” to control pain. To some extent, this approach was based on a “labeled line” theory of pain sensation; that is, there are tracts and central nervous system (CNS) centers that are specific to nociception, and that by interrupting these sites, analgesia can be achieved. That this worked at all is a tribute to our early understandings of nociception.

As we have come to understand the nociceptive system, it is clear that pain perception is a highly complex phenomenon, and that there are no “pain centers” in the brain that can be destroyed or removed, akin to resection of an epileptogenic focus to alleviate a seizure disorder. Despite this, a completely nihilistic approach to destructive operations for pain control is unwarranted.

Anterolateral cordotomy is probably the best example of the power of disruptive surgery to achieve pain relief. During the mid-20th century, open surgical cordotomy proved to be an important method of surgical pain control, and the procedure became even more practical when the technique of percutaneous cordotomy was developed and popularized. Attaining somatic analgesia from cancer pain caudal and contralateral to a spinal cord lesion, using a technique that could be performed without incision, was clearly a breakthrough. As Drs. Cleary and Cetas point out, similar success for noncancer pain has never been routinely achieved, although interest in minimally invasive interruption of pathways and nuclei for these pains does remain.

Likewise, dorsal root entry zone (DREZ) lesions proved highly effective for pain syndromes in which dorsal roots had been traumatically avulsed from the spinal cord, often when a phantom pain was experienced. This was perhaps the first, and last, example of durable analgesia from a destructive lesion in the CNS for noncancer pain. In this case pain relief from DREZ appears to be durable.

A further successful example of a destructive surgical approach to pain is trigeminal rhizotomy. This can be performed on the peripheral trigeminal branches by open surgical technique, percutaneous lesion by radiofrequency thermal injury, glycerol injection or balloon compression, radiosurgery, or open surgical rhizotomy by a posterior fossa approach. In this case, it is probably the triggering stimulus that is affected, rather than the pain mechanism itself, and the results are often not permanent.

Apart from these few examples, predictable pain relief from surgical disruption of primary afferents, deep nuclei (medial thalamus), or neocortex (cingulate gyrus) has been elusive. The same can be said for sympathectomy, which seems to have almost disappeared from the pain surgeon's toolbox.

Each destructive procedure for pain control is unique, and requires both experience and skill. My concern is that a generation of neurosurgeons have been exposed to few such procedures, sometimes to none of them. The knowledge and expected outcomes of our mentors and teachers are receding on the horizon. Unless we identify these procedures and subject them to study, by contemporary standards of evidence, we risk losing valuable insights and approaches that can benefit patients with cancer and noncancer pain. My hope is that the next generation of pain surgeons will take up this cause and will convincingly demonstrate what works and what does not work—certainly for cancer pain, and particularly for neuropathic pain.

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21

Facet Blocks and Denervations

Sunil J. Panchal and Allan J. Belzberg

Each spinal segment from C2 caudal possesses three joints: anteriorly, the disk and associated uncovertebral joint; and posteriorly, the paired facet joints. For almost a century, the lumbar facet (zygapophyseal) joint has been considered a significant source of low back pain. Ghormley was the first to describe the *facet syndrome*, which he defined as a lumbosacral pain with or without radiculopathy, occurring most often after a sudden twisting or rotary strain of the lumbosacral region.¹ Hirsch et al. injected hypertonic saline in the region of the lumbar facet joints, which resulted in pain in the sacroiliac and gluteal regions with radiation to the greater trochanter.² Mooney and Robertson performed saline intraarticular facet injections that resulted in a similar pain referral pattern; however, they noted that the pain was relieved by intra-articular local anesthetic injection.³ Similar findings were produced in the cervical spine. Cervical facet injection with hypertonic saline by Pawl resulted in neck pain and headache.⁴

Although the “slipped disk” has commanded the spotlight as the principal cause of low back pain, surgical removal of the disk usually does not afford relief from axial back pain. A spinal fusion, which stops the motion of the facet joint, often is required for adequate control of back pain. The pathophysiology of low back pain is a complex issue, with various soft tissues and bony structures of the spine vying for the distinction of pain generator. Among these, the facet joint likely plays a significant role.

■ Anatomy

Facet Joints

The facet joints are paired diarthrodial synovial joints formed by the inferior articular process of one vertebra and the superior articular process of the vertebra below.⁵ They are present from the C1–C2 junction to the L5–S1 junction. A tough fibrous capsule is present on the posterolateral aspect of the joint. There is no fibrous capsule on the ventral aspect of the facet joint. Instead, the ligamentum flavum is located ventrally, in direct contact with the synovial membrane. Adipose tissue surrounding the spinal nerve is in direct contact with adipose tissue located in the superior recess of the facet joint, allowing direct spread of injectate from the joint to the epidural space and potentially to the spinal nerve.^{6,7} The capacity of the joint space averages only 1.0 to 2.0 mL in total volume. Communications between ipsilateral or contralateral facet joints do occur, often via defects in the pars interarticularis. These account for some of the spread of anesthetic that can occur during facet injections.

Facet Innervation

Each spinal nerve root divides into a posterior and an anterior ramus. The *posterior ramus*, also known as the *sinuvertebral* nerve of von Luschka, divides approximately 5 mm from its origin into *medial*, *lateral*, and *intermediate* branches. In turn, the medial branch divides into two branches that supply both the facet joint at the same level and the joint at the level below.⁸ Therefore, each joint has a dual innervation supply (**Fig. 21.1**). The location of the medial branch and its divisions vary from the lumbar, cervical, and thoracic regions in relation to the bony structures.

In the lumbar region, the medial branch is located in a groove at the base of the superior articular facet, where it crosses the transverse process posteriorly and inferiorly. It then divides, sending a branch medially and cephalad to the joint at the same level and a branch inferiorly to the joint below. The medial branch also supplies the multifidus and interspinalis muscles as well as the ligaments and periosteum of the neural arch.² Therefore, neural blockade of the medial branch

is not specific for facet joint pain. There is some evidence of joint innervation from a third ascending branch, which originates directly from the mixed spinal nerve (**Fig. 21.1**).^{5,9,10}

Innervation of the cervical facet region differs in that the medial branch predominantly supplies the facet joints, with minimal innervation of the posterior neck muscles.¹¹ The C3–C4 to C7–T1 facet joints are supplied by the medial branches from the same level and the level above.^{12,13} These branches wrap around the waist of each articular pillar bound to periosteum by investing fascia and the tendons of the semispinalis capitis.⁴ The medial branch of C8 crosses the T1 transverse process, similar to the orientation of lumbar facet innervation. The C3 medial branch divides earlier in its course into a deep, superficial (third occipital nerve) branch. The deep C3 medial branch descends to innervate the C3–C4 facet joint; the superficial medial branch (third occipital nerve) traverses the lateral and dorsal surface of the C2–C3 facet joint before entering the joint capsule.^{11,13,14} The atlantooccipital and lateral atlantoaxial joints receive innervation from the C1 and C2 ventral rami (**Fig. 21.2**).

The thoracic facet joint innervation has a pattern similar to that of the lumbar region, except for the T5–T8 levels. The medial branches at these

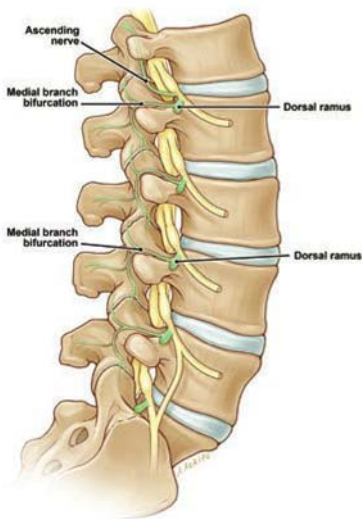


Fig. 21.1 Illustration of the lateral view of the lumbar spine with corresponding facet joint dual innervation patterns of the dorsal rami. The medial branch indicated demonstrates bifurcation after crossing the transverse process, with one branch terminating in the inferior pole of the adjacent facet joint, and the other branch descending to the superior pole of the facet joint below. Additionally, a third ascending nerve directly from the spinal root provides innervation to the facet joint above.

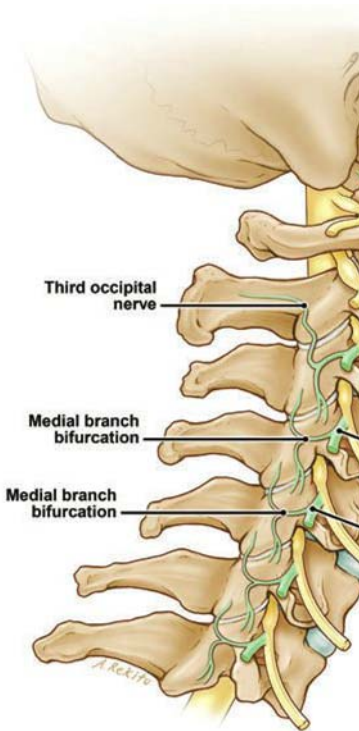


Fig. 21.2 Illustration of the lateral view of the cervical spine with corresponding facet innervation patterns. Each medial branch courses along the lateral surface of each articular pillar, and then bifurcates for a dual innervation pattern for the facet joints. Also shown is the location of the atlantooccipital joint, atlantoaxial joint, the C2 ventral ramus, and the third occipital nerve.

levels travel lateral from the foramen, cross the superior lateral border of the transverse process, and course medial to innervate the corresponding facet joint and level below.¹⁵

■ Pathophysiology

Intervertebral disk space narrowing occurs as the disk degenerates and loses hydration. The change in segment height can cause subluxation of the facet joints, resulting in abnormal stresses on the joint and nerve root impingement. Other sequelae, such as capsular irritation and local inflammation, may result in reflex spasm of the erector spinae muscles. As degeneration proceeds, abnormal motion leads to osteophyte production, exacerbating the symptoms.¹⁶

That the facet joint is a source of nociception has yet to be universally accepted. Opponents submit that local anesthetic blockade of the facet joint with subsequent pain relief lacks validity. This position is supported by observations of contrast injection spilling over into the epidural space or intervertebral foramen.⁷ Pain elicited with hypertonic saline or relief with local anesthetic administration may be due to action on neural structures or on other pain-sensitive tissues.

Proponents for the facet joint as a site of nociception point to the presence of substance P in facet capsule neurons.¹⁷ In addition, most of the mech-

anosensitive somatosensory units in the facet joint are group III high-threshold, slow-conduction units, which are thought to mediate nociception.^{18–20} Chronic inflammation may lead to fluid accumulation and distension, stimulating the richly innervated synovial villi inside the capsule, resulting in pain.

■ Facet Block: Diagnostic or Therapeutic Tool?

Lumbar facet arthropathy is characterized by low back pain, unilateral or bilateral, with or without radiation. The pain is described usually as a deep, dull ache;

is difficult to localize; and frequently is referred into the buttock, groin, hip, or posterior thigh to the knee (**Fig. 21.3**). Fukui et al. described referral patterns for thoracic facet joints.²¹

Some patients describe a sudden onset of pain, usually associated with twisting or bending. There is no exacerbation of the pain with Valsalva maneuver. In contrast to discogenic pain, sitting does not severely aggravate pain secondary to facet arthropathy.

The cervical facet joints also cause pain described as deep and aching. Referral patterns vary, depending on which level is of concern. The C1–C2 facet joint may refer pain to the occipital and postauricular region.²² The C2–C3 facet joint may cause pain referred to the occiput, ear, vertex, forehead, or eye.^{23,24} The C3–C4 facet joint refers pain over the posterolateral cervical region, following the course of the levator scapulae. The lower cervical facet joints refer pain to the base of the neck and down to the scapulae (**Fig. 21.4**).²³

Physical examination often reveals tenderness over the facet joints and involves associated muscle spasm. The pain is exacerbated by extension or lateral bending as opposed to flexion as well as prolonged sitting. A few patients may exhibit mechanical hyperalgesia over the associated innervated skin. Although range of motion in all directions may be reduced, extension and rotation are most uncomfortable. Straight leg raise is usually negative.

To make the diagnosis of a painful facet joint requires the typical history and physical findings already described in combination with diagnostic blocks. The use of facet block for diagnosis is hampered by certain pitfalls. There is a lack of a corresponding cutaneous innervation to the facet joint and thus an inability to determine when complete blockade has occurred. Injection into the joint often results in joint capsule rupture and spillage of local anesthetic into the epidural space or intervertebral foramen, which can interrupt nociceptive impulses from alternative sites.^{20,25–27} The medial branch nerve innervates muscles, ligaments, and periosteum in addition to the facet joints, again limiting specificity of the test. Facet blocks should be avoided in

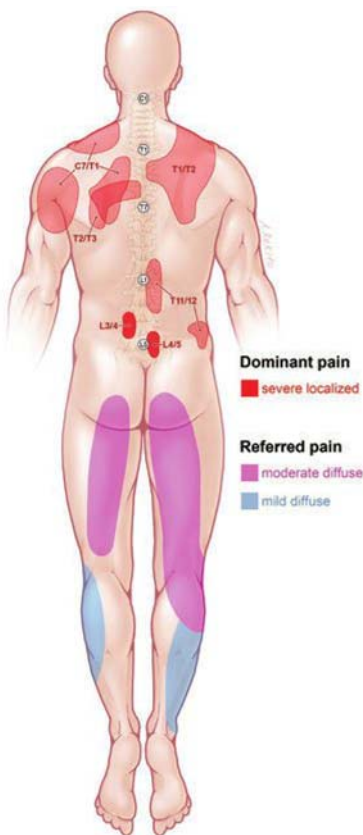


Fig. 21.3 A composite map of distribution of thoracic and lumbar facet syndrome pain, from volunteers who had undergone intra-articular needle placement.

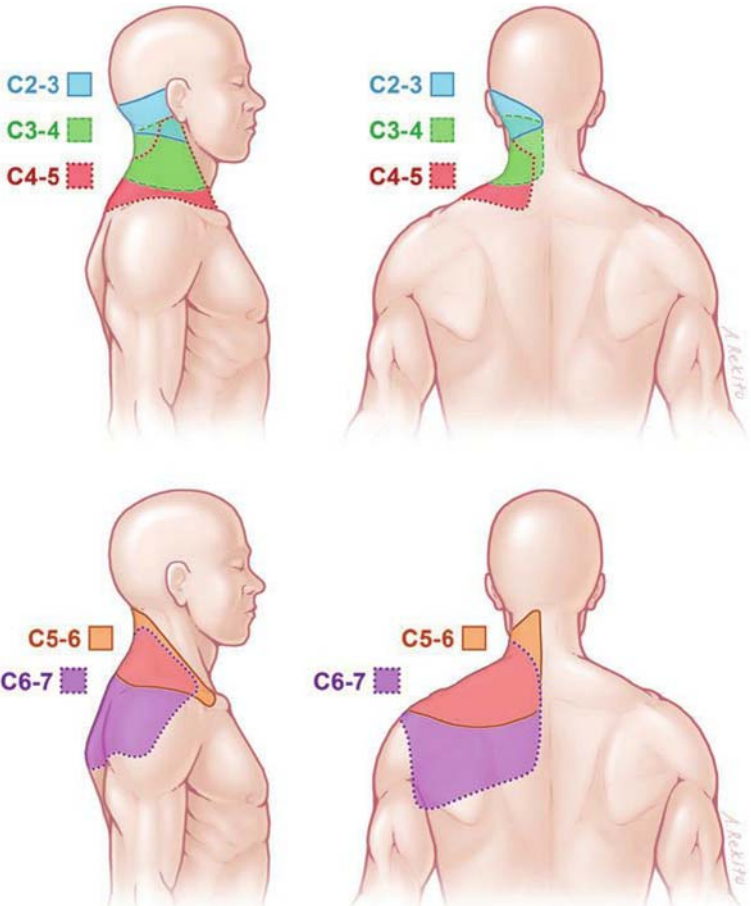


Fig. 21.4 A composite map of cervical facet syndrome pain referral patterns after intra-articular needle placement.

patients with systemic infection, infection at the site, or coagulopathies, or in patients who refuse the procedure.

Needle placement for facet injection as well as local anesthetic delivery can result in pain provocation. A provocative response that is concordant with the patient's ongoing complaints lends further support to the notion that the facet joint is the pain generator.

Facet injections are commonly used for both therapeutic and diagnostic interventions. Intra-articular steroid injection often produces significant pain relief that outlasts the action of a local anesthetic.^{26,28-30} Although therapeutic benefit from steroid has been demonstrated, long-term outcomes have been disappointing.³¹⁻³³ Intra-articular block also does not correlate well with the success of

radiofrequency (RF) denervation (only 64%); therefore, medial branch block is the preferred procedure as a trial prior to facet denervation.³⁴

■ Technique

Lumbar and Thoracic Facet Blocks

For facet joint injection, the patient is positioned prone, with an abdominal cushion to reduce lumbar lordosis. Sterile preparation and draping of the back are performed. Intra-articular injection requires oblique fluoroscopic views. Best results are achieved at a 30- to 45-degree plane to “open” the joint. Either the table or the C-arm can be rotated for optimal viewing. The entry point through the skin then is identified and marked with the aid of a radiopaque instrument. The skin is infiltrated with 1% lidocaine using a 25-gauge needle. A 22-gauge, 3.5-inch spinal needle then is introduced via the skin wheal and advanced into the joint using a trajectory parallel to the fluoroscopy beam. Local anesthetic alone or with steroid (0.25% bupivacaine and 20 mg Depo-Medrol [methylprednisolone acetate]) is delivered in a volume of 1.0 to 1.5 mL. Volumes in excess of 2 mL will rupture the capsule and spill over into the epidural space.

For medial branch block, the patient is positioned prone, and the transverse process for each branch to be blocked is identified using fluoroscopy. Approximately 5 cm from the midline, a skin wheal is raised, and a 22-gauge 3.5-inch spinal needle is advanced to the medial end of the transverse process, contacting the dorsal surface of the process near the superior edge. The L5–S1 medial branch is blocked at the groove between the ala of the sacrum and the superior articular process of the sacrum (**Fig. 21.1**). A total volume of 1.0 mL of 0.5% bupivacaine is delivered at each site, and the patient is questioned for concordance compared with the original pattern of referred pain. For the T5–T8 levels, the ideal site of placement is the superolateral aspect of the transverse processes.

The patient is ideally positioned prone in order to reduce potential injury to the vertebral arteries, but lateral position as well as supine for the upper levels have been described. Sterile technique and needles are used as previously outlined. Needles are introduced 1 to 2 cm lateral to the waist of the articular pillar, guided by a posteroanterior view on fluoroscopy. The needle then is advanced to the centroid of the articular pillar as seen on a lateral view. Again, 1.0 mL of local anesthetic is delivered (**Fig. 21.5**).

Intra-articular injection at the cervical level is not favored for several reasons. Cervical joint spaces are small and narrow. Further, the epidural space is immediately medial to the joint, and the vertebral artery is just lateral to the joint. Therefore, direct injection into cerebral circulation or blockade of cervical nerve roots is of great concern.

Facet Joint Denervation

Denervation of the medial branch can be accomplished by using either radiofrequency ablation or cryoneurolysis. Each method is described here in terms of its mechanism of action, followed by the results of long-term outcome studies.

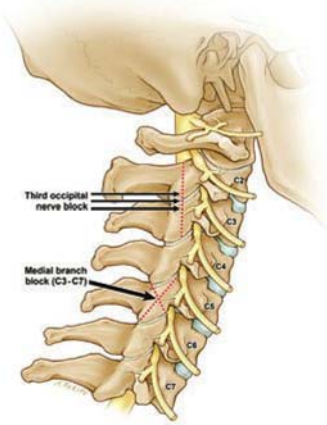


Fig. 21.5 Illustration of a lateral view of the cervical spine demonstrating correct needle placement for medial branch blocks at levels C3–C7. The desired target is the centroid of the articular pillar as seen at the intersection of the superimposed dotted lines. Also, the third occipital nerve for block of the C2–C3 facet joint is targeted at three positions (arrows). The *upper* arrow is above the subchondral plate of the inferior articular facet of C2, the *middle* arrow is over the joint line, and the *lower* arrow is below the subchondral plate of the superior articular facet of C3.

Conventional Radiofrequency Ablation

The radiofrequency lesion generator has the following critical functions: (1) continuous online impedance measurement; (2) nerve stimulation; (3) monitoring of voltage, current, and wattage during radiofrequency lesioning; and (4) temperature monitoring. Electric impedance is measured to confirm the continuity of the electric circuit and to detect short circuits. Impedance is usually 300 to 600 ohms in extradural tissue. The nerve stimulator is used to detect proximity to sensory or motor fibers of the segmental root. Stimulation at 50 Hz is used to detect sensory fibers; 2 Hz is used to detect motor fiber stimulation. Ford et al. demonstrated that if the electrode is resting on the nerve, 0.25 V will be required to produce discharge, whereas 2 V will be required to produce discharge at a distance of 1 cm.³⁵ Therefore, monitoring voltage is important in determining proximity.

Temperature monitoring occurs at the tip of the electrode only, with a thermocouple technique, producing a thermodynamic voltage that is proportional to temperature.

Bogduk et al. performed lesions in egg whites and meat and found that radiofrequency lesions do not extend

distal to the electrode tip. Instead, lesions extended radially around the electrode tip in the shape of an oblate spheroid with a maximal effective radius of 2 mm using a 21-gauge electrode with a 3-mm exposed tip.³⁶ **Table 21.1** demonstrates a survey of varying tip sizes and temperatures with the corresponding lesion sizes. The first visible signs of coagulation occur at 62°C, but neural destruction begins at 45°C. The maximal lesion size is attained once the “working” temperature is maintained for 20 to 40 seconds. Maintaining the temperature for longer periods did not result in any discernible increase in lesion size.³⁷ Although initial reports indicated selectivity for small fibers, Uematsu conclusively showed that radiofrequency indiscriminately damages both small and large fibers.³⁸

Retrospective studies demonstrate similar success rates of lumbar denervation. Goupille et al. and others showed a 38.4% success rate at 2 years, and North et al. showed a 45% success rate with a mean follow-up of 3.2 years.^{39–41}

Table 21.1 Survey of tip sizes and temperatures

Investigators	Electrode diameter (mm)	Exposed electrode tip length (mm)	Tip temperature (°C)	Transverse lesion size (mm)	Test medium
Cosman et al. 1988 ⁷⁵	216	3	65	2–4	Egg
Bogduk et al. 1987 ³⁶	186	5	80	2.2 ± 0.4	Egg
	226	4	80	1.1 ± 0.2	Egg
	226	4	90	1.6 ± 0.2	Egg
Moringlane et al 1989 ⁷⁶	216	2	60	3.7	RC
	216	2	70	5.5	RC
	216	2	80	7.2	RC
Vinas et al. 1992 ³⁷	206	4	80	4.9	RC

Abbreviation: RC, rabbit cortex.

North et al. went further, concluding that there was no difference in success for bilateral denervation for bilateral pain compared with unilateral denervation for unilateral pain.⁴¹ Goupille et al. reported that patients who did not have prior spine surgery had better success with denervation, whereas North's group did not show any statistical difference (**Table 21.2**). Van Kleef et al. performed a lumbar facet RF denervation double-blinded randomized, controlled trial (RCT) in 31 patients with 80 C lesions at L3–L4, L4–L5, and L5–S1 with a sham control. At 8 weeks, the mean visual analog scale (VAS) score was 4.8 for controls and 2.8 for the treated group. This was statistically significant for both differences in VAS, and for Oswestry scores as well. In the treated group, 10 of 15 patients were successfully treated (at least a 2-point reduction on VAS and greater than 50%

Table 21.2 Long-term outcome of radiofrequency (RF) denervation

Investigator	Type	No. of patients	Length of follow-up	Success (>50% relief)	Failure	Outcome with history of previous back surgery
Goupille et al. 1993 ⁴⁰ (RF)	R	10	24 mo	38.4%	61.6%	Worse
North et al. 1994 ⁴¹ (RF)	R	42	Mean, 3.2 y	45%	55%	No difference

Abbreviation: R, retrospective.

pain relief) at 8 weeks, and of these patients, 7 were still a success at 12 months.⁴² Nath et al. performed a sham-controlled RCT of lumbar facet RF denervation in 40 patients after at least 80% pain relief was documented from controlled medial branch blocks. The RF group had multiple lesions performed at each level. At 6 months, the RF group had statistically significant improvement in VAS scores and in the patients' global assessment in comparison with the sham group. There was also significant improvement in secondary measures such as spine range of motion, quality-of-life measures, and physical exam findings posttreatment.⁴³ A 10-year prospective clinical audit of lumbar facet RF denervation in 209 patients was able to maintain 2-year follow-up data on 174 of the patients. Of these individuals, 119 (68.4%) had good (>50%) to excellent (>80%) relief at 6 months. At 12 months, 81 patients still had good to excellent relief, and this was maintained in 36 patients at 24 months.⁴⁴ See **Table 21.2**.

Stolker et al. reported on 40 patients who underwent thoracic facet denervation with a mean follow-up of 31 months.⁴⁵ They found that 44% were pain free and 39% had greater than 50% relief. Stolker and coworkers also performed a cadaver study, with fluoroscopic guidance, in which radiofrequency denervations were performed bilaterally at T1–T12. They found that 61% of the lesions hit neural tissue, but *none* hit the medial branch stem (the “target”).⁴⁶

The nerve stimulator should be used in an attempt to reproduce the patient's usual pain complaints and achieve better localization of the thoracic medial branch.

A randomized, double-blind trial of 24 patients with cervical facet pain after a motor vehicle accident was performed to compare percutaneous radiofrequency denervation of multiple lesions at 80°C with controls. Patients were selected for study after confirmation of cervical facet syndrome by use of double-blinded, placebo-controlled diagnostic local anesthetic blocks. Follow-up assessment was performed to determine the time until pain returned to 50% of the preprocedural level.

Radiofrequency patients had a median duration of relief of 263 days compared with 8 days in the control group.⁴⁷ In a separate study, psychological distress was measured by the McGill Pain Questionnaire and the SCL-90-R psychological questionnaire in patients with whiplash injury. A significant resolution of psychological distress was associated with pain relief from cervical facet radiofrequency denervation.⁴⁸

A prospective study was performed to assess for differences in outcomes of cervical facet RF denervation for treatment of whiplash symptoms based on litigation status. Patients with pain that persisted after 20 weeks were referred for RF treatment and followed for 1 year ($N = 46$). There was significant improvement in pain immediately after treatment and at 1 year follow-up, but no statistical difference between litigants and nonlitigants. Pain scores for nonlitigants were reduced by 2.0 immediately and by 2.9 at 1 year, and by 2.5 and 4.0, respectively, for litigants.⁴⁹

In regard to recurrence of pain and repeat treatment, two reports of small (20 and 24 patients) retrospective studies of repeat procedures after successful RF were identified for cervical and lumbar facet denervation. In both series, more than 80% of patients had > 50% relief from repeat RF treatment, and mean duration of relief from subsequent RF treatments was comparable to the initial treatment.^{50,51}

Pulsed Radiofrequency Ablation

Pulsed radiofrequency (PRF) treatments involve the application of short pulses of RF energy to neural tissue ranging from 5 to 50 ms with a frequency ranging from 1 to 10 Hz. The most common setting described is 2 Hz and 20 ms, with the goal of keeping the tissue temperature below the denaturation threshold of 45°C. This method has been theorized to be nonablative, and to provide relief by inducing intracellular changes, but has not been determined to have either of these benefits in a definitive manner.^{52–54} In vitro studies suggest that PRF may change morphology of mitochondria and alter axonal structures, and it has been demonstrated to reduce neuropathic pain behavior in the rat Chung model as well as a sciatic nerve ligation study in rabbits.^{54,55} The clinical experience for utilization of PRF for lumbar facet pain has been positive, but PRF does not appear to enjoy the same duration of effect as conventional RF. Tekin et al. performed a randomized trial of PRF versus conventional RF, with similar rates of improvement at 6 months, but only the RF group had maintained benefit at 1 year.⁵⁶ Van Zundert et al. randomized 23 patients to PRF versus sham treatment and results were better immediately, but not significantly different at 6 months.⁵⁷

Cryoanalgesia

The application of cold temperatures for pain relief has been used for centuries. Trendelenburg noted that freezing caused nerve damage with loss of function, but regeneration always occurred without formation of scar or neuroma. Neural damage from freezing is possibly secondary to the hypertonicity of intracellular and extracellular fluids, physical damage from ice crystals, cell protein damage, or ischemic necrosis.^{58–63} If freezing occurs slowly, crystals form mostly in the extracellular space, and neurons may not be completely injured. If freezing occurs rapidly, large intracellular crystals form and destroy the cell. The critical temperature for cell injury is –4°C or lower, but may be variable. Below –20°C, cells are irreversibly damaged. Slow thawing leads to recrystallization (smaller crystals melt, whereas large crystals grow temporarily) and greater cellular damage.⁶⁴ Also, the longer the freeze time, the greater the diameter of the ice lesion.

Cryolysis probes function based on the Joule–Thompson effect (**Fig. 21.6**), which states that gas under pressure emitted from a small orifice will expand and with expansion cools significantly. Probes are made of stainless steel with a coaxial design. Those with thermocouples allow control of the target temperature. The probe tip may be either hemispherical or shaped like a needle bevel. A hemispherical tip will create a circular ice lesion with the tip at the center, whereas the beveled tip will create an ice lesion proximal to the tip. Large (14-gauge) probes have electrical nerve stimulation and temperature-monitoring capability, but smaller probes do not. With the use of N₂O or CO₂, one can achieve a temperature of –50°C to –70°C at the tip. The temperature gradient from the probe tip will change 10°C/mL of distance. It has been noted that lesion size can be increased by freeze–thaw cycles.⁵⁵ Brechner demonstrated that cryoprobe application for facet block did not result in a significant change in the temperature of cerebrospinal fluid (CSF) (**Table 21.3**).⁶⁶

Histopathologic analysis reveals that, after cryolysis, wallerian degeneration occurs, but the perineurium and epineurium remain intact. Duration of relief is dependent on the distance the axon must cross during regeneration. Regrowth averages 1 to 3 mm per day.^{67,68}

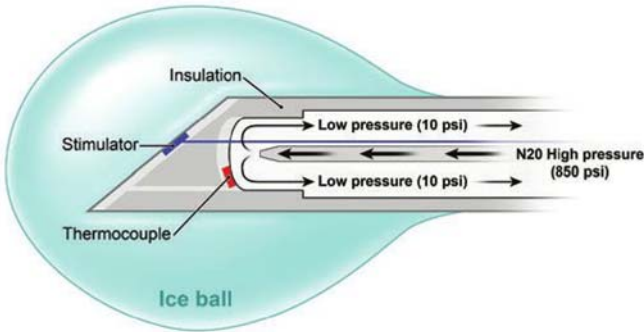


Fig. 21.6 Illustration of a cryoprobe with electrical stimulation capabilities. The probe is insulated except at the distal end, with a coaxial shaft designed to circulate N₂O under high pressure, which then expands as it enters the larger outer canal. Expansion of the gas leads to a decrease in temperature, which is measured by the thermocouple. N₂O, nitrous oxide; psi, pounds of pressure per square inch.

The success rate of cryolysis is optimistic. Ross et al. reported on 23 patients who underwent medial branch cryoneurolysis for facet syndrome; these patients had follow-up of 6 months to 2 years. During this time, 2 patients had recurrence of pain (at 6 and 8 months) and had good response to repeat treatment.⁶⁹ Schuster monitored 52 patients over 13 months. Significant relief was reported by 47 patients, and only 1 patient had recurrence requiring repeat denervation, which occurred at 9 months.¹⁰

■ Complications

Complications from facet block are infrequent and transient. A brief exacerbation of pain may occur and last a few days to a few weeks. Intrathecal injection has been reported, as well as one case of chemical meningitis.⁷⁰ Epidural blockade has occurred, and vertebral artery puncture and strokes have been described at the cervical level.

Radiofrequency denervation resulted in postprocedure pain in 13% of patients in one study; the pain resolved spontaneously over 2 to 6 weeks. No persistent motor or sensory deficits were reported.

Table 21.3 Temperature changes (°C) at various sites with cryoprobe at ventral surface lamina

	Control	Postfreeze
Intrathecal	37.5	36.4
Dorsal surface lamina	36.8	36.4
Ventral surface lamina	36.5	28.9

■ Systematic Literature Reviews

A 2007 systematic review of facet joint interventions utilizing AHRQ (Agency for Healthcare Research and Quality) criteria found that the evidence for pain relief with RF denervation is moderate for short- and long-term pain relief at the cervical and lumbar levels, but was indeterminate for thoracic facets.⁷¹ A 2009 systematic review of diagnostic utility and therapeutic effectiveness of cervical facet joint interventions by Falco et al. found level II-1 or II-2 evidence (controlled trials without randomization, and cohort or case control studies from more than one center) for RF neurotomy in the cervical spine using U.S. Preventive Services Task Force (USPSTF) quality ratings.⁷² Using the same rating system, Datta and colleagues found level II-2 and level II-3 (cohort or case control studies from more than one center, and multiple time series with or without the intervention) evidence for lumbar radiofrequency neurotomy.⁷³ Van Boxem and colleagues, in a review of evidence for continuous and pulsed RF, note that RF at the cervical and lumbar levels has produced the most solid evidence, and differences in outcome among RCTs can be attributed to differences in patient selection and/or inappropriate technique.⁷⁴

■ Conclusion

The facet joints are increasingly accepted as a source of axial spine pain with common referral patterns, but methods for diagnosis remain underutilized. Specificity of local anesthetic injection is limited, but clearly medial branch block is

Editor's Comments

The data to support facet denervation is modest and the benefits short-lived, probably measured in months at best. Drs. Panchal and Belzberg present the anatomical case for facet rhizolysis. What we do not have is an explanation of why these procedures fail so quickly. The short-term relief in 50% of patients may well fall within the placebo response range. In fact, it is known that the original Shealy lumbar facet rhizotomy knives were of inadequate length to access the sinuvertebral nerve, yet the procedure was still effective in many cases.

It is likely that the multilevel nature of the innervation of the facet joints is in part to blame. It is also likely that there are multiple anatomical contributors to neck and back pain: Anterior and posterior longitudinal ligaments, the interspinous ligament, the dura, the annulus fibrosus, putative pathologic innervation of the degenerated nucleus pulposus, and the paraspinal musculature are all possible candidates. Segmental localization of neck and back pain is also notoriously difficult, particularly in patients with multilevel disk degeneration and osteoarthritis.

To some extent, the odds are against the procedure from the outset. We still lack the diagnostic specificity and precision to diagnose the origin of neck and back pain. It is possible that even this goal is a “fool’s errand,” and it may be that *all* the aforementioned structures involved in the spinal motion segment contribute to pain when the segment has degenerative change. To attack just one of the constituents is almost certain to fail.

As pointed out in this chapter, one of the most effective and durable methods to address axial pain is spinal fusion, yet this drastic step cannot be effectively tested preoperatively, and cannot be reversed. It is unfortunate that this major procedure has supplanted more minor palliative procedures like facet denervation.

preferred compared with intra-articular injection when attempting to prognosticate relief from denervation.

Local anesthetic injections as well as denervation, with either radiofrequency or cryoneurolysis, are performed easily, are well tolerated by patients, and are extremely safe. Pain relief can be achieved in about 50% of patients for a reasonable duration.

Directions for future study include investigation of outcomes of thoracic facet RF denervation, and for patients with multiple areas of degenerative changes, outcomes of combined denervation treatments across targets are desirable.

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22

Dorsal Rhizotomy and Dorsal Root Ganglionectomy

Feridun Acar and Selçuk Göçmen

From the beginning of the 20th century, destructive procedures targeting different locations in the pain pathway have been used.^{1,2} Despite their value and efficacy, newer neuromodulation techniques and current medications have diminished in prominence as approaches in the surgical management of pain. However, in some cases, more contemporary neuromodulation approaches are inadequate to control a particular pain. Moreover, neuromodulation techniques are typically expensive and need both follow-up and maintenance. With this perspective, a pain surgeon should know the indications for destructive procedures and skills to perform these procedures.^{3,4}

Otfried Foerster was the pioneer who described dorsal rhizotomy to control spasticity.⁵ In 1966 Scoville described the technique of spinal dorsal root sectioning,⁶ and in 1970 Smith⁷ reported combined dorsal rhizotomy and sectioning of sympathetic rami communicans. In 1974, Uematsu reported on percutaneous radiofrequency rhizotomy.⁸ Osgood et al⁹ introduced dorsal root ganglionectomy in 1976.

■ Anatomical Background

Each spinal nerve is attached to the spinal medulla by two roots: posterior (afferent) and anterior (efferent) (**Fig. 22.1**). The posterior root is larger than the anterior root because it contains a larger number of radicular fibers and the individual fibers are of larger size. The posterior rootlets have a vertical linear attachment to the posterolateral sulcus of the spinal medulla. The fibers of the posterior rootlets are in close proximity and in some instances overlap. The posterior root separates as it passes away from the spinal medulla into two bundles, both of which become connected with the proximal end of a spinal ganglion. From the distal end of this ganglion, the posterior root proceeds to its junction with the anterior root in the intervertebral foramen. The spinal ganglia are found on the posterior roots of all the spinal nerves. In the case of the first cervical segment the spinal ganglion may be rudimentary or absent and the posterior root itself may be derived from the accessory nerve. The posterior roots occupy the intervertebral foramina except in the case of the sacral and coccygeal nerves, where the ganglia lie within the vertebral canal, and the first and second cervical nerves, where the ganglia lie upon the vertebral arches of the atlas and axis, respectively. The ganglia are of ovoid form and bifurcated in some cases at the proximal end.¹⁰

The anterior root arises from the anterior surface of the medulla in the anterior root exit zone by means of bundles of nerve fibers, which occupy a greater horizontal area and are more irregular in their arrangement than the radicular

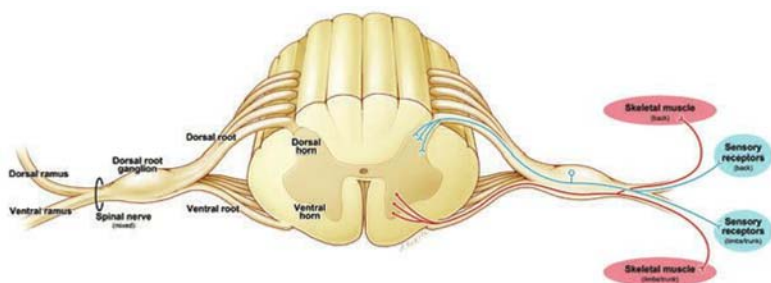


Fig. 22.1 The anatomical organization of the spinal nerve. Each spinal nerve is attached to the spinal medulla by two roots: posterior (afferent) and anterior (efferent). The afferent fibers carry the sensory input and efferent fibers innervate the skeletal muscles.

fibers of the posterior root. It possesses no ganglion in its course. The nerve fibers sometimes overlap and are sometimes connected with neighboring radicular fibers above and below.¹⁰

From their attachment to the spinal medulla the dorsal and ventral roots proceed laterally in the vertebral canal toward the intervertebral foramina, where they unite to form the spinal nerve. The direction of the first two cervical spinal nerve rootlets is superior and lateral; the rootlets of the remaining spinal nerves course obliquely inferiorly and laterally, the obliquity gradually increasing from the cervical area to the inferior conus medullaris.

The dorsal and ventral rami of the spinal nerves contain fibers from both posterior and anterior roots. Indeed, each root can be seen on removal of its sheath to divide into two portions, one of which enters into the formation of the dorsal ramus and the other into the formation of the ventral ramus. With the exception of the first two spinal nerves, the dorsal rami are uniformly smaller than the ventral rami.

■ Indications for Dorsal Rhizotomy and Ganglionectomy

The indications for dorsal rhizotomy and ganglionectomy can be divided into two broad categories: those pains related to cancer and those pains unrelated to cancer. Both procedures produce irreversible changes of the peripheral and central nervous systems. Thus, the evaluation of the patient prior to surgery is paramount. A detailed clinical workup will help the physician to determine the proper level and the number of roots and ganglia to be sectioned. The determination of level can vary between a single root or ganglion and multilevel procedures. The level of surgery also depends on intradural root anastomosis and the rich interplay of the sympathetic system and ventral root sensory fibers.

Preoperative local anesthetic blocks can be helpful in determining the levels for surgery. However, diagnostic blocks can also be misleading due to the block

technique or to a placebo effect. The outcome of diagnostic blocks is one factor in surgical decision making, and cannot be used as the sole determinant.

Cancer Pain

In patients with malignancies involving the brachial plexus such as invasive breast carcinoma, multilevel cervical dorsal rhizotomy to denervate a functionally impaired limb can be successful in controlling pain.¹¹ In patients with intact upper extremity motor function, this procedure can result in severe functional loss of the involved limb.

In patients with malignancies of thorax and chest wall, thoracic ganglionectomy and rhizotomy can be good surgical options.¹¹ There is a segmental overlap of dermatomes in the thoracic area that mandates multilevel rhizotomy or dorsal root ganglionectomy in these patients. A three- to five-level ganglionectomy and/or rhizotomy centered on the target dermatome(s) should be considered to completely denervate one or two segments.

A successful outcome can be achieved with dorsal rhizotomies in cases with pain secondary to pelvic neoplasms.^{9,12-14} The most important disadvantage of this surgery is the destruction of S2 and S3 sensory innervation, which will lead to neurogenic bladder, neurogenic bowel, and also impotence.

Noncancer Pain

The most common indication for ganglionectomy and rhizotomy in noncancer pain is the treatment of occipital neuralgia.¹⁵⁻¹⁷ Occipital neuralgia can be successfully treated with C2 and C3 ganglionectomies.¹⁵⁻¹⁷ C2 and C3 ganglionectomies can also be considered for medically resistant cervicogenic headache.

In patients with pain syndromes presenting with allodynia, ganglionectomy of the corresponding level can be considered. The allodynia-dominant pain is often seen in postthoracotomy, postlaparotomy, and postherpetic pain syndromes.^{3,17-19}

■ Technique

Cervical Dorsal Rhizotomy and Ganglionectomy

Intradural cervical rhizotomy is rarely performed, but can be achieved by identifying the roots by their exiting foramina. The roots can be cut dorsal to the denticulate ligament to preserve the anterior rootlets. For C2 ganglionectomy, the ganglion is exposed through a midline incision between the inion and the transverse process of C3, and dissection of the paravertebral musculature to expose the C1–C2 interspace is completed.²⁰ The venous complex surrounding the root and ganglion is cauterized dorsally using bipolar electrocautery. The root exiting from the C2 space is identified and followed distally until the ganglion is identified. The nerve should be cut just proximal to the ganglion, allowing it to retract slightly, and the proximal stump cauterized to prevent bleeding. The nerve is then followed distally and removed (**Fig. 22.2**). The C3 ganglionectomy is performed in a similar fashion with the addition of a foraminotomy to expose the C3 nerve root and ganglion (**Fig. 22.3**). Once the ganglion is identified, it is removed as in C2 surgery.

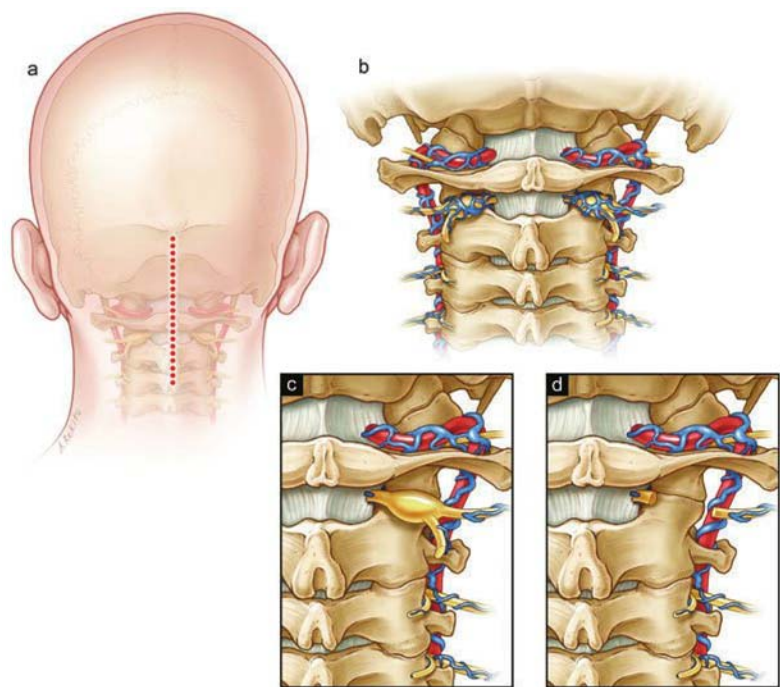


Fig. 22.2 (a–d) Illustration of C2 ganglion. No foraminotomy is needed to expose the ganglion. Note that the ganglion is covered by venous plexus.

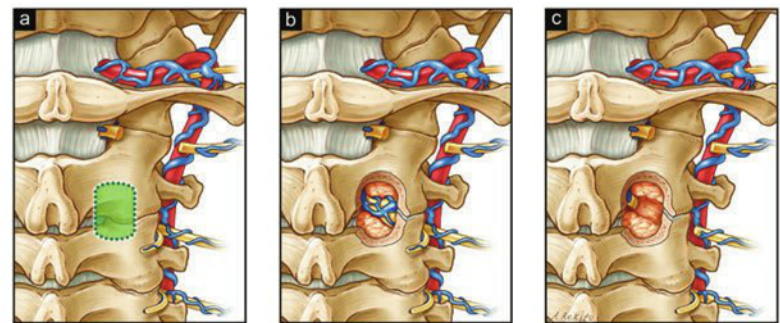


Fig. 22.3 (a–c) Illustration of C3 ganglion. Note that a foraminotomy is needed to expose the ganglion. This ganglion is also covered by venous plexus. Careful coagulation is necessary before the ganglion excision.

Thoracic Dorsal Rhizotomy and Ganglionectomy

Dorsal rhizotomy can be performed through an intradural or extradural approach. The dorsal rhizotomy is performed similarly to cervical rhizotomy. The larger feeding segmental arteries that can enter along the posterior roots must be protected.²¹ A multilevel laminectomy is a standard for intradural rhizotomy. In general, the roots of (at least) three segments should be destroyed to accommodate overlap innervations.²² In this approach, the correct sensory roots are identified by opening dura at their respective intervertebral foramina. They then are cut after coagulating. The procedure for extradural rhizotomy is comparable to that of extradural ganglionectomy without resection of the ganglion.

A thoracic ganglionectomy may be used to relieve the thoracic neuropathic pain, such as intercostal neuralgia. The patient is placed in a prone position under general anesthesia and the relevant transverse process is confirmed by fluoroscopy. After midline incision, the paravertebral muscles are displaced from the spinous process and from the laminae and the lateral edge of the facet joints until the transverse processes are exposed. The surgeon can perform a small lateral bone resection of the laminae, lateral facetectomy, and foraminotomy with a small Kerrison rongeur or a high-speed drill. Under a surgical microscope, the dural cuff of the root is identified and followed through a foraminotomy, exposing the root in both directions, and the ganglion identified. The dorsal and motor roots usually are enclosed within the same dural sheath, but occasionally are contained within separate dural sheaths. The feeding segmental arteries that pass through the foramen should be protected. The dural cuff is exposed further laterally to the bifurcation of the spinal nerve, exposing the intercostal nerve originating from the ventral branch and allowing identification of the radiculomedullary artery originating from the intercostal artery. A proximal ligature of the proximal sensory root is necessary to prevent CSF (cerebrospinal fluid) leakage. The ganglion is grasped, elevated, and bluntly dissected off the fibrous septum. Distally, the ganglion is cut after coagulating and sectioning its distal connection to the spinal nerve.¹¹

A posterior paraspinal approach may be used for a radiofrequency rhizotomy in the thoracic region. The theory behind radiofrequency lesions of the dorsal roots and dorsal root ganglia was developed by Letcher and Goldring,²³ who noted that the action potentials of nociceptive fibers (A δ and C fibers) are blocked at lower temperatures compared with the larger tactile fibers (A α and A β fibers). With fluoroscopic guidance a 2-mm uninsulated-tip electrode is placed within the neural foramen. Proper placement is confirmed and sensory stimulation is then performed at less than 1 V with 50-Hz stimulation to ascertain paresthesia in the region of the patient's pain. Motor stimulation is then tested at 2 Hz to elicit contraction in the paraspinal muscles. A test lesion is made at 42°C for 15 seconds. A permanent radiofrequency lesioning is performed at 65 to 90°C for 60 to 90 seconds.¹¹

Lumbar intradural dorsal rhizotomy is performed similarly to cervical dorsal rhizotomy. In the sacral region, Crue and Todd reported on the technique of extradural rhizotomy.¹² In brief, the S1 and S2 roots are exposed after a bilateral upper sacral laminectomy. The thecal sac is then ligated with 0-silk suture. One tie is passed around the thecal sac just caudal to the S1 nerve root axilla, and a second tie is passed around thecal sac just rostral to the S2 nerve root axilla. The thecal sac is sharply cut with its contents between the two ties, preserving the S1 nerve roots.

For lumbar ganglionectomy, the ganglion is exposed within the intervertebral foramen. Resection of the lateral margin of the facet joint and the medial aspect of the inferolateral margin of the pars interarticularis is required. For S1 ganglionectomy,²⁴ the standard L5–S1 hemilaminectomy and foraminotomy are performed. After the dural sheath of the S1 root is identified, the dorsal root is sectioned proximal and distal to the ganglion. The ganglion is elevated, and bluntly dissected off the fibrous septum, which separates it from the ventral motor root. Finally, the root is ligated proximally, and then the ganglion is cut distally.

■ Outcome

Despite the fact that the history of destructive procedures for the treatment of pain dates back to the beginning of modern neurosurgery, there are relatively few clinical papers in peer-reviewed journals that contain detailed descriptions of long-term outcomes of dorsal rhizotomy or dorsal root ganglionectomy, employing validated outcome measures and standardized follow-up.

Only a few studies meet Class I evidentiary criteria. Cetas et al. reviewed 146 articles about destructive procedures for the treatment of nonmalignant pain.²⁵ In general, these studies showed either a modest treatment effect (in the case of radiofrequency neurotomy for facet pain), consistent with earlier case series, or no effect (in the case of ganglionectomies), despite the positive results reported from uncontrolled studies.

Published studies are confounded by both reporting and observer bias. The subjective nature of pain and the frequent absence of an objective treatment effect marker further complicate the assessment of the literature on this topic.²⁵ Almost all of the reviewed studies demonstrated an early, dramatic response to treatment that tended to deteriorate over long-term follow-up.²⁶

Dorsal Rhizotomy

A total of 24 dorsal rhizotomy studies have been published concerning spinal and other pain syndromes (**Table 22.1**). In the majority of the studies (14 of 24) the data reported were relevant to lumbar facet syndromes (LFS) (**Table 22.1**, first section; 14 studies).^{4,6,22,27–37,48} The remainder examined the effects of rhizotomy on a variety of truncal and extremity pains (**Table 22.1**, third section; 9 studies).^{38–46}

The effects of dorsal root rhizotomies on a variety of localized pain syndromes were published between 1966 and 1973.^{4,6,22,29,32} Echols²⁹ and White and Kjellberg⁴ reported a success rate (variably defined) of just over 60% in two reports on 62 patients. Loeser,²² Scoville,⁶ and Onofrio and Campa³² reported encouraging early success rates, but long-term results were modest (25–50%). Despite the variability in the pain syndromes treated, number of sectioned roots, outcome measures, and follow-up, the results were apparently discouraging enough that the procedure was effectively abandoned and replaced with modified rhizotomies directed at facet denervation for the treatment of facet syndromes.²²

Radiofrequency rhizotomies of the medial branch of the dorsal root were compared with sham or intrafacetal versus extrafacetal rhizotomies in blinded RCTs (randomized controlled trials).²⁵ Outcome measures and patient selection differed sufficiently between the groups to preclude a meta-analysis.⁴⁷ The treated group

Table 22.1 Literature review of rhizotomy: grouped according to disorder treated

Lumbosacral pain					
Author(s) and year	Study design	Diagnosis	No. of patients	Follow-up	Outcome
Bärlocher et al. 2003 ²⁷	Case series	LFS	50	1 y	1-y VAS scores and work capacity assessment: 62% w/ good response, 38% not successful
Dreyfuss et al. 2000 ²⁸	Prospective audit	LFS	15	1 y	~ 60% w/ 90% relief of pain at 12 mo, 87% w/ at least 60% relief
Echols 1969 ²⁹	Case series	Chronic UE and LE pain after ≥ 1 ops for ruptured intervertebral disk	62	Not stated	40% w/ op failure, 60% dramatically and probably permanently relieved of pain by section of one or two sensory roots
Gallagher et al. 1994 ³⁰	RCT	LFS	41	6 mo	At 1- and 6-mo FU, statistical difference between placebo and rhizotomy (33% VAS score reduction at 1 mo; 13% at 6 mo)
Leclaire et al. 2001 ³¹	Prospective double-blind RCT	LFS	70 pts w/ LBP lasting >3 mo w/ good response after intra-articular facet injections under fluoro (36 pts w/ perc RF articular facet denervation under fluoro, 34 w/o denervation [placebo])	4 and 12 wk	At 4 wk, Roland–Morris score improved by a mean of 8% in neurotomy group and 2.2% in placebo group; Oswestry or VAS scores not significant per tx; at 12 wk no tx effect based on the three scales
Loeser 1972 ²²	Case series	Intractable LBP	33 NM/46 total	3 mo–10 y	63% w/ overall initial success, 28% w/ overall long-term success; 29 pts w/ initial excellent/good results, but 19 w/ long-term failure

(Continued)

Table 22.1 (continued) Literature review of rhizotomy: grouped according to disorder treated

Lumbosacral pain					
Author(s) and year	Study design	Diagnosis	No. of patients	Follow-up	Outcome
Pevsner et al. 2003 ³³	Case series	Mechanical back pain	122	18 mo	12-mo FU: 63% w/ good results, 37% w/ no effect; 18-mo FU of 22 pts: all had significant pain relief; 22% w/ minor complications
Onofrio and Campa 1972 ³²	Case series	Intractable LBP	286 pts total, 218 available for FU	Not stated	25–50% relief for group w/ pain of unknown origin, other groups not stated directly (“many groups had no success”)
Sanders and Zuurmond 1997 ³⁴	Randomized blinded?	LFS	34	3 mo	VAS and Oswestry Disability Index used: intrafacet injections better than extrafacet
Scoville 1966 ⁶	Case series	Various	10 NM/12 total	Not stated	40% w/ good results
Staender et al. 2005 ³⁵	Case series	LFS	76	Median 22.5 mo	VAS score 6.7 preop and 2.9, 3.2, and 3.4 at 3 d, 3 mo, and 6 mo postop, respectively; in 40% of pts, pain reduced for ≥ 12 mo
Tzaan and Tasker 2000 ³⁶	Case series	LFS	118	Mean 5.6 mo	41% had >50% pain reduction
van Kleef et al. 1999 ³⁷	RCT/prospective double-blind	LFS	31	12 mo	8 wk postop, tx successful in 10 pts in tx group and 6 in control group; unadjusted OR 3.3 ($p = 0.05$, NS) and adjusted OR 4.8 ($p < 0.05$, significant); differences in effect on the VAS scores, global perceived effect, and Oswestry Disability Index were statistically significant; and at 3, 6, and 12 mo postop there were significantly more tx successes in pts in the RFL compared w/ the sham group
White and Kjellberg 1973 ⁴	Case series	Mixed	62	Unclear (1 mo–17 y?)	Overall 64.5% w/ “permanent success”
Oh and Shim 2004 ⁴⁸	RCT	Chronic discogenic LBP	49	4 mo	VAS significantly lower in lesion group

Cervical pain					
Author(s) and year	Study design	Diagnosis	No. of patients	Follow-up	Outcome
Barnsley 2005 ³⁸	Case series	Cervical zygapophysial joint pain	35	2 y	36 of 45 ops achieved significant pain relief, w/ mean duration of pain relief of 36 wk
Govind et al. 2003 ³⁹	Prospective trial	3rd occipital HA	49	90–900 days	88% w/ initial successful outcome, median duration of relief 297 d
Lord et al. 1996 ⁴⁰	Randomized double-blind trial	Pain in ≥ 1 cervical zygapophysial joints after auto accident	24 pts: 12 w/ perc RF neurotomy w/ multiple lesions and temperature of lesion-making electrode raised to 80°C; 12 w/ identical op except RF current off	263 d	Median elapsed time before pain returned to at least 50% of preop level: 263 d in active tx and 8 d in control group ($p = 0.04$); at 27 wk, 7 pts in active tx and 1 in control group were free of pain
McDonald et al. 1999 ⁴¹	Case series	Cervical zygapophysial joint pain	28	Up to 730 d	Complete pain relief in 71% of pts
Sapir and Gorup 2001 ⁴²	Case series	Cervical whiplash	46	1 y	Significant overall reduction in cervical whiplash symptoms and VAS score post tx and at 1 y
Stovner et al. 2004 ⁴³	Randomized double-blind trial	Cervicogenic HA	12 pts: 6 w/ RF neurotomy of facet joints C2–C6	2 y	No difference after 3 mo

(Continued)

Table 22.1 (continued) Literature review of rhizotomy: grouped according to disorder treated

Cervical pain					
Author(s) and year	Study design	Diagnosis	No. of patients	Follow-up	Outcome
van Kleef et al. 1996 ⁴⁴	Prospective double-blind randomized trial	Intractable chronic cervico-brachial pain	20 pts: Group I, 67°C RFL adjacent to DRG, and Group II, no RFL	8 wk	Significant no. of successfully treated pts in Group I compared w/ Group II ($p = 0.0027$); significant reduction in VAS score ($p < 0.01$) and also in parameters measured w/ MPQ-DLV and MPI-DLV in Group I—therefore, 67°C RFL adjacent to DRG can result in a significant alleviation of chronic cervicobrachial pain
van Suijlekom et al. 1998 ⁴⁵	Case series	Cervicogenic HA	15	16.8 mo	VAS and VRS used; RF neurotomy of cervical zygapophysial joints significantly reduced HA severity in 80% of pts at short- and long-term assessment
Wallis et al. 1997 ⁴⁶	Randomized double-blind trial	Cervical zygapophysial joint pain	17	3 mo	

Abbreviations: DRG, dorsal root ganglia; fluoro, fluoroscopy; FU, follow-up; HA, headache; LBP, low back pain; LE, lower extremity; LFS, lumbar facet syndrome; MPI-DLV, Multidimensional Pain Inventory, Dutch Language Version; MPQ-DLV, McGill Pain Questionnaire, Dutch Language Version; NM, nonmalignant; NS, not significant; OR, odds ratio; perc, percutaneous; RCT, randomized controlled trial; pts, patients; RF, radiofrequency; RFL, RF lesioning; tx, treatment; UE, upper extremity; VAS, verbal analog scale; VRS, 7-point verbal rating scale.

showed modestly improved pain scores in the long term. They found that the intrafascial rhizotomies were superior to extrafascial procedures.²⁵ Complications were minimal in all blinded RCTs. Patients with “good outcomes” varied from 41 to 75% in long-term follow-up of prospective studies. The success rates for Class III studies ranged from 40 to 60% at long term (see **Table 22.1**, first section).²⁵

Oh and Shim evaluated the effects of treatment of chronic discogenic back pain using radiofrequency lesions of the ramus communicans.⁴⁸ After a failed intradiscal electrothermal procedure, patients were screened using a good response by local nerve blockade of the ramus communicans as an endpoint. Patients with other identifiable causes of back pain, such as lumbar facet syndrome and radiculopathy, were excluded. At 1 and 4 months of follow-up, the treated group showed significant improvement on the VAS and the 36-Item Short Form Health Survey (SF-36).

Radiofrequency rhizotomies were compared with sham procedures for the treatment of cervical pain^{40,44} or cervicogenic headache.⁴³ Patients were carefully selected based on their response to repeated nerve blocks with local anesthetics and followed for a period from 8 weeks to 3 years. Two of the RCTs demonstrated a significant effect of the radiofrequency lesions,^{40,44} although one study did not.⁴³ Based on a single Class II study, a pain score reduction was durable.³⁹ One study failed to demonstrate any effect, but was underpowered.⁴³ Both studies with long-term follow-up demonstrated similar median times to recurrence^{39,40} (**Table 22.1**, third section).

Dorsal Root Ganglionectomy

A total of 17 articles related to ganglionectomy studies. Two of these studies were RCTs. In one,⁴⁹ radiofrequency lumbar ganglionectomy was compared with sham surgery for sciatica, and in the other, radiofrequency lesions made at two different temperatures were compared.⁵⁰ The remaining articles were Class III case series ranging in size from 3 to 102 patients (**Table 22.2**).^{7,15,16,19,34,49–61}

Geurts et al. reported on 83 patients who had either radiofrequency lesion (45 patients) or needle placement without radiofrequency lesion (38 patients) of a selected lumbar ganglion.⁴⁹ All the cases were adult patients with clear radicular pain symptoms. Patients were further selected by their response to repeated local nerve blocks. No statistical difference was found between groups. Slappendel et al. also found no difference in outcomes between brachialgia patients receiving radiofrequency lesions directed at the ganglion.⁵⁰

Jansen reported the efficacy of C2 ganglionectomies for occipital neuralgia.⁵³ Eighty percent of patients had significant relief of symptoms, but long-term follow-up data were less compelling. The next largest series of patients compared ganglionectomies performed in patients with occipital pain described as either “sharp, burning, jabbing, electrical, or exploding” (Group I) or “dull, aching, throbbing, or pressurelike” (Group II).⁵⁵ Patients in Group I had a higher prevalence of a traumatic history (74%) and had the best response: nearly 80% improved. Overall, Group II patients had a poor response. Ganglionectomy articles are listed in **Table 22.2**.

Acar et al. performed ganglionectomy procedures in 20 patients (50% men) with occipital neuralgia.²⁰ Pain was localized to the C2, C3, or both distributions in 8 (40%), 9 (45%), and 3 patients (15%), respectively. They studied three groups of patients: C2 ganglionectomy (4 patients), C3 ganglionectomy (5 patients), and

Table 22.2 Literature review of ganglionectomy

Author(s) and year	Study design	Diagnosis	No. of patients	Follow-up	Outcome
Dubuisson 1995 ¹⁵	Case series	Occipital neuralgia	11	Unclear	71% w/ likelihood of improvement
Fedder 1990 ⁵¹	Case report	Minor causalgia	4	6–12 mo	All w/ relief and decreased narcotic use
Geurts et al. 2003 ⁴⁹	Multicenter double-blind RCT	Chronic lumbosacral radicular pain	83 total: 45 w/ RFL, 38 control	3 mo	At 3 mo, 16% of treated pts and 25% of control had successful tx; lumbosacral RFL of DRG showed no advantage over control tx w/ local anesthetics
Hosobuchi 1980 ⁵²	Case series	Intractable chest pain	3	3 y	2 of 3 developed total anesthesia, including loss of dysesthesia, hyperesthesia, and original pain
Jansen 2000 ⁵³	Case series	Cervicogenic HA	102	Unclear	80% improved
Kato et al. 1990 ⁵⁴	Case report	Neuropathic pain	3	1–32 mo	1 w/ complete pain relief, 2 w/ partial pain relief
Lozano et al. 1998 ⁵⁵	Case series	Medically refractory chronic occipital pain	39	19–48 mo	19 pts w/ excellent results; pts w/ pain from trauma or Group I criteria responded best to ganglionectomy (80% good or excellent response), but pts w/ nontraumatic pain or Group II did not have favorable results
Murphy 1969 ⁵⁶	Case series	Occipital neuralgia of various origins	30	< 1 y–5 y	18 w/ excellent; 7 w/ good, 3 w/ fair, 2 w/ poor results (unclear what criteria were used to determine outcome measurements)
North et al. 1991 ⁵⁷	Case series	Failed back surgery syndrome	13	Mean 5.5 y	2 w/ success at 2 y postop, 0 w/ success at 5.5 y postop; 1 w/ equivocal success at 2 y postop, 2 w/ equivocal success at 5.5 y postop
Osgood et al. 1976 ⁵⁸	Case series	Lumbosacral pain, BPA, amputation	18	Mean 7 mo	10 w/ “good” results, 4 w/ fair

Sanders and Zuurmond 1997 ³⁴	Case series	CHA	66	Mean 29.1 mo (Group A), 24 mo (Group B)	intermittent CHA (Group A): 60.7% w/ complete relief; chronic CHA (Group B): 30% (3 of 10) w/ complete relief
Slappendel et al. 1997 ⁵⁰	Prospective double-blinded RCT	Cervicobrachialgia	Group 1 (32 pts): RFL at 67°C; Group 2 (29 pts, control): RFL at 40°C	3 mo	identical outcome in two groups
Smith 1970 ⁷	Case series	Postthoracotomy, herpes	10 NM/12 total	Unclear	Per authors: "all had some degree of relief"
Stechison and Mullin 1994 ¹⁶	Case series	Greater occipital neuralgia	5	Mean 24 mo	5 w/ immediate postop relief of pain; 1 w/ recurrence of pain at 26 mo underwent ganglionectomy; all w/ nausea and dizziness postop, and 1 w/ a CSF leak
Taub et al. 1995 ⁵⁹	Case series	Sciatica	61	< 1 y–15 y	59% w/ pain markedly reduced or eliminated
Wetzel et al. 1997 ⁶⁰	Case series	Postop chronic lumbar radiculopathy	51	6 mo, 2 y	55% good or excellent at 6 mo, the rest w/ poor or failed outcomes; at final FU (2-y minimum) 19% of 37 pts w/ good or excellent outcome
Wilkinson and Chan 2001 ⁶¹	Retrospective chart review	Various origins	19	Mean 22 mo	74% reported > 50% reduction, only 1 pain free

Abbreviations: BPA, brachial plexus avulsion; CSF, cerebrospinal fluid; CHA, cluster headache; DRG, dorsal root ganglia; FU, follow-up; HA, headache; NM, nonmalignant; pts, patients; RCT, randomized controlled trial; RFL, radiofrequency lesioning; tx, treatment.

C2 and C3 ganglionectomies (11 patients). Consecutive C2 and C3, or C3 and C2 ganglionectomies were performed due to failure of an initial single-level ganglionectomy. Although 95% of patients had significant relief from pain, the long-term follow-up results showed excellent relief (pain free) in only 4 patients (20%), moderate relief (50–90% relief) in 8 (40%), and poor relief (<50% relief) in 8 (40%). There was no statistically significant predictor of favorable outcome. Diagnostic ganglion blocks had strong correlation with short-term favorable outcomes ($p < 0.005$) but no influence on long-term pain relief ($p > 0.5$).

■ Conclusion

Destructive surgeries such as ganglionectomy and rhizotomy may still have a role in the treatment of some pain syndromes mentioned in this chapter. Destructive procedures for the treatment of pain have played an important role in the history of neurosurgery, but their efficacy has not been well established based on contemporary standards.

How should future studies be designed and conducted? The answer is clear: RCTs (single blinded) in patients with standardized and well-documented diagnoses should be conducted. Well-designed studies with appropriate control groups will be necessary to provide evidence that meets contemporary standards.

Editor's Comments

Dorsal rhizotomy is one of the oldest procedures performed for pain relief. As with many historical procedures, the contemporary evidence to support its use needs to be strengthened.

Doctors Acar and Göçmen have described the surgical technique at length. Although it appears simple, the procedure can be quite daunting, particularly at C2 or C3, given the venous plexus that surrounds the root and ganglion. In my experience, time devoted to bipolar coagulation of this plexus, and its careful dissection, saves considerable time and blood loss.

The present chapter is an excellent review of the current state of the evidence on the topic of dorsal rhizotomy and dorsal root ganglionectomy. The procedures are straightforward, and it should certainly be possible to conduct randomized crossover trials that compare surgical and nonsurgical therapy over a reasonable follow-up period. Long-term follow-up of the same study group could answer the question of how durable pain relief is after surgery. Indications can also be refined in the design of such a study.

The authors do not discuss the theoretical advantage of dorsal root ganglionectomy over dorsal rhizotomy, given the presence of “ventral root afferents,” which are known to overrepresent small myelinated and unmyelinated axons. Removing the ganglion would disrupt all pain fibers, whereas dorsal rhizotomy would spare only ventral root afferents. There has been no high-quality trial of dorsal root ganglionectomy since these afferents were discovered more than three decades ago.

In my opinion, neurosurgeons will continue to use these procedures in the future for selected indications, and it is important that our trainees gain some familiarity with these operations. It will be up to those of us who practice surgical pain management to generate the data that support or reject this modality for long-term pain control.

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Percutaneous Computed Tomography–Guided Cordotomy for Pain

Ahmed M. Raslan and Ashwin Viswanathan

Anterolateral cordotomy is a spinothalamic tractotomy. It was one of the first central nervous system (CNS) ablative procedures to be done to control pain in humans.¹ The term itself was coined by Schüller.²

Cordotomy has a history that in itself tells the story of pain surgery over the last century. When cordotomy was introduced, it was a very popular procedure and a frequently performed operation up until the 1970s and early 1980s. The early iteration was an open procedure that involved a laminectomy and sectioning of the anterolateral cord, with an associated morbidity and mortality.

In the 1960s, a second iteration of the procedure was introduced that was percutaneous. Several destructive methods were tried, and eventually experts settled on a radiofrequency (RF) thermolysis.^{3,4} This iteration was a significant improvement on open cordotomy because it eliminated much of the morbidity related to open surgery. The percutaneous procedure was widely used for pain control for both cancer-related and noncancer-related pain.

By the early 1980s oral opioids, in many formulations, were being widely used for the control of pain. This, and a general aversion to surgical procedures for pain, led to a significant reduction in the number of cordotomies being performed. In that environment, a third iteration of cordotomy was developed by Kanpolat⁵ in Turkey. This cordotomy, whereas still percutaneous, introduced computed tomography (CT) as a method of direct visualization of the radiofrequency electrode within the spinal cord prior to and during the radiofrequency lesion—an addition that would render the procedure safer and more effective. CT-guided cordotomy did not gain traction in North America initially but was employed in other parts of the world, particularly those areas where the technology of intrathecal drug delivery was cost prohibitive. Only recently has there been a renewed interest in cordotomy in North America. It is this most advanced form of cordotomy, and refinements of this technology,⁶ that is the focus of this chapter.

■ Anatomy

The spinothalamic tract carries nociceptive signals, temperature, and nondiscriminative touch from the contralateral side of the body. The spinothalamic tract axons migrate ventrally as they ascend the length of the spinal cord. In segments rostral to the cervical enlargement, axons do not migrate farther ventrally but continue in a position ventral to that in which they ascend through thoracic segments.⁷ There is a somatotopic organization of axons within the spinothalamic tract; fibers entering from rostral and caudal segments are located in the medial and lateral parts of the tract, respectively. The spinothalamic tract terminates mainly in the ventro-

posterolateral nucleus, the ventroposteromedial nucleus, the intralaminar nuclei (mainly the central lateral nucleus), and the posterior complex. Spinothalamic tract projections to the central lateral nucleus of the thalamus play a part in the motivational-affective responses to pain, and the projection to the lateral thalamus (the ventrobasal complex) is involved in sensory-discriminative aspects of pain.⁸ The corticospinal tract lies posterior to the lateral spinothalamic tract (LST) with white matter (the safety zone) in between (**Fig. 23.1**). The ventral spinocerebellar tract overlies the LST and a lesion that eliminates the spinocerebellar tract may cause ipsilateral ataxia of the arm. Human autonomic pathways for vasomotor and genitourinary control in addition to the reticulospinal tract that controls ipsilateral automatic respiration are also part of the anterolateral quadrant of the spinal cord. Therefore, sleep apnea (Ondine curse), incontinence, and hypotension are possible undesirable cordotomy effects. There is considerable variation in the size and location of the ventral corticospinal tract; absence of decussation is also possible. Motor decussation may extend from the obex to the C1 level; contralateral leg weakness may also occur if a lesion is too high.

■ Candidacy

The indications for cordotomy have also evolved over time. Early in its history it was used mainly for pain unrelated to cancer. Over time, it has come to be used primarily for cancer-related pain. The ideal candidate is a cancer patient with unilateral somatic pain with a life expectancy of under a year. Bilateral pain is more difficult to treat, and midline pain generally does not respond to cordotomy. Further, pain above the dermatome of C5 is generally not well controlled by cordotomy. Pulmonary function tests demonstrating an forced expiratory volume at one second (FVC1) and forced expiratory volume (FEV) > 50% are generally the minimal standards for the procedure.⁹

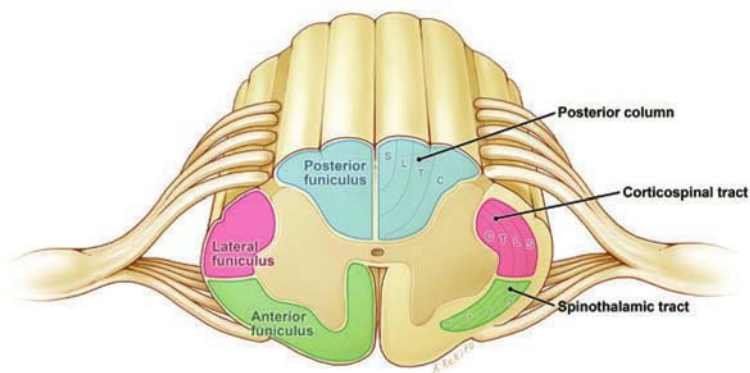


Fig. 23.1 Somatotopic organization of spinothalamic and corticospinal tracts in the cervical spinal cord.

Pain due to mesothelioma represents the prototypic cancer-related pain that is responsive to cordotomy given its unilateral nature and the somatic type of pain resulting from involvement of the pleura and ribs. Pulmonary involvement is usually a late development in mesothelioma, as is impairment of pulmonary functions beyond the minimal cutoff.¹⁰

■ Evidence

The evidence for cordotomy stands almost alone among other ablative spinal cord procedures. It is the most studied and has the highest number of dedicated publications and the highest number of patients reported, and is second only to sympathectomy in terms of the quality of data.

In a recent structured review of all percutaneous ablative procedures, 3,601 patients were reported in 47 publications between 1966 and 2010; many reports involved more than 200 patients, with follow-up exceeding 6 months.^{3,11–15} The most recent cordotomy literature has originated outside the United States, where the economic environment has favored ablative over neuromodulation procedures. There were no Class I reports of cordotomy found in this review. One prospective trial was identified.¹⁶ This trial used standardized outcome measures: the visual analog scale (VAS), the Karnofsky performance scale (KPS), activities of daily living (ADL), and total sleeping hours. The author reported a statistically significant improvement in all outcome measures that compared postprocedure and baseline pain levels. Three reports were retrospective cohorts with survival analysis of pain relief of the entire cohort until death.^{17–19} Many other reports were retrospective cohorts with large numbers (> 100) of patients and 6 months of follow-up.

GRADE is a system that separates the strength of recommendation from the underlying quality of evidence. When we applied the GRADE system of evidence to cordotomy, it received a 1C grade—a strong recommendation for the use of cordotomy in cancer pain, but based on a low level of evidence.²⁰

■ Technique

Equipment

The needle, stylet, and electrode we use is the KCTE kit provided for CT-guided spinal cord radiofrequency lesioning (Cosman Medical, Burlington, MA, USA). A RF lesion generator that is capable of stimulation and impedance monitoring is needed.

The procedure is generally performed in the CT scanner in the radiology suite; however, with the recent availability of wide-bore mobile intraoperative CT scanners, the procedure may also be done in the operating theater.

Preoperative Preparation

Patients are usually admitted the same day. Anticoagulant or antiplatelet agents are stopped at least 1 week prior to the procedure.

Thirty minutes prior to the procedure, a lumbar injection of 12 mL of Omnipaque 300 mg/mL (GE Healthcare, Princeton, NJ, USA) is performed and patients are then kept in the Trendelenburg position.

Surgical Positioning

Cordotomy is done in the supine position and the head is immobilized in the CT gantry. It is important to maintain the head in a straight, neutral position to facilitate the three-dimensional orientation during needle placement. The shoulders are pulled down to ensure adequate access to the side of the neck to allow placement of the electrodes. The area around the mastoid tip is prepped and draped.

Imaging and Data Acquisition

Parallel, preferably zero-gantry-angle, 1.0– to 1.25-mm cuts are scanned spanning from the foramen magnum down to the bottom of C2 with a wide field of view (FOV) to allow imaging of the skin. The FOV can be adjusted to avoid dental artifact. The cut of choice to perform the procedure is between C1 and C2, where there is a lateral space to allow introduction of the needle. The laser beam of the scanner is used to guide the entry point into the skin. The skin dura thickness and the anteroposterior and lateral diameter of the cord are all determined from the slice through which skin entry will be accomplished (**Fig. 23.2**).

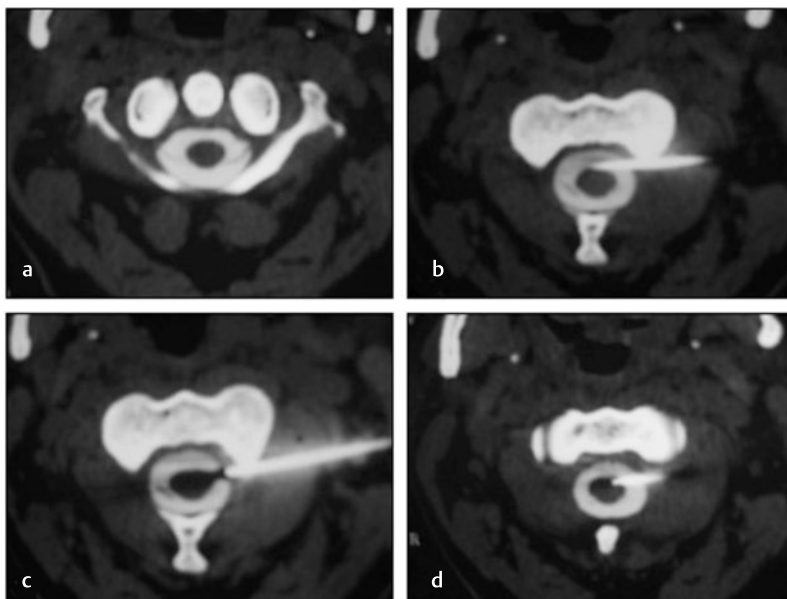


Fig. 23.2 (a) CT (computed tomography) myelography revealing the dentate ligament and the upper cervical spinal cord; the cord diameter and skin dura thickness are obtained from the image. (b) The needle introduced to a trajectory anterior to the cord. (c) The needle trajectory is adjusted to proper targeting of the anterolateral quadrant. (d) The electrode is inserted through the needle into the spinal cord.

Needle Placement

After local infiltration with lidocaine 2%, the cannula of the KCTE kit is introduced through the skin to a length that is shorter than the skin dura thickness in a direction mostly perpendicular to the skin at the level of the mastoid tip in the anteroposterior plane. CT is used frequently to check the position of the cannula until the dura is punctured. Before puncture of the dura 2 mL of xylocaine 2% can be injected to avoid pain due to contact with the sensitive C2 ganglion and dura. After free flow of CSF (cerebrospinal fluid) is obtained, a CT check is always performed, then according to the previously determined lateral diameter of the cord, the length of exposed tip of the electrode is adjusted on the hub of the electrode and the electrode is introduced through the cannula. Cord penetration is signaled by a sudden increase in the impedance of the CSF values (< 400 ohms) to approximately 1,000 ohms. Typically at this point the patient experiences a mild pain and/or electric sensation.

Target Verification

Radiological

By CT, the position of the electrode is confirmed in relation to the geometry of the cord (the opposite anterolateral quadrant of the cord in relation to the side of pain). The electrode placement can also be confirmed through the relation of the area of maximal pain intensity and the somatotropic arrangement within the spinothalamic tract: more anteromedial for the arm, and more posterolateral for the leg.

Clinical

Usually cord penetration produces a brief somatotopic-specific pain, which can also be confirmed by stimulation of the electrode.

Electrophysiological

Impedance Monitoring

Impedance monitoring is useful in identifying the degree of cord penetration only, and not the location of the electrode within the spinal cord.

Macrostimulation

The electrodes we use for lesioning allow us to stimulate the spinothalamic tract (the target) in both high (sensory) and low (motor) frequencies to verify the targeting.

All patients are stimulated with 100-Hz pulses of duration 0.1 ms, producing a sense of warmth and/or painful sensation in the opposite half of the body at < 0.15 V, covering the painful region. Motor stimulation at 2 Hz and pulse duration 0.1 ms is then performed up to 1.0 V. There should be no motor response in the extremities with stimulation.

Lesion Making

We begin with one test lesion (60 s, 60°C) and then check the contralateral half of the body for hypoesthesia and any pain relief. The test lesion is completed with the ipsilateral leg of the patient elevated off the table to detect any subtle change of the motor power during the lesion process. If adequate hypoesthesia or pain relief is not obtained from the test lesion (which is the case in most patients), a second lesion at 75°C for 90 seconds is performed.

The benchmark of a successful lesion is the abolition of the patient's ability to differentiate a sharp pin prick and a dull sensation. Once a successful lesion is accomplished, the cannula is removed from the patient and the puncture site is dressed.

Postoperative Care

Overnight observation with continuous pulse oximetry is needed for cordotomy patients to monitor for possible sleep apnea. Opioids can be weaned on the first postoperative day, but in most cases, this is deferred to a week after the procedure.

■ Results

Since cordotomy is rarely done for non–cancer-related pain, the results of cordotomy in this group are from older studies and are generally inferior to the results of cordotomy for cancer-related pain.²¹ However, this general notion has been challenged by case reports of prolonged pain relief after cordotomy for non–cancer-related pain.⁶

Table 23.1 summarizes the results of percutaneous cordotomy in cases of cancer-related pain published in English between 1966 and 2010.^{3,11–15} The results aggregate all cordotomies published, only a small fraction of which were done using CT guidance. CT-guided cordotomy is categorically different from fluoroscopic cordotomy because CT allows direct visualization of target-electrode relationship, leading to higher safety and precision. In a series of 41 patients,¹⁶ the immediate postoperative results of CT-guided cordotomy ranged between 92% and 98% pain relief, or satisfactory pain relief, declining to 80% at 6 months, with a persistent clinically and statistically significant reduction in the VAS (**Fig. 23.3**). Kanpolat et al also reported an initial success of 97%, which declined to 84% at 6 months' follow-up.²²

■ Complications

There have been no mortalities reported in the literature for CT-guided cordotomy, although a 6.25% mortality rate has been reported for the older fluoroscopic cordotomy.⁵⁶

Ondine curse can result from cordotomy when pulmonary function is impaired or the only functioning lung is the lung ipsilateral to the cordotomy

(text continued on page 304)

Table 23.1 Results of percutaneous cordotomy for cancer pain from 1966 to 2010 in all English-published peer-reviewed manuscripts, redundancy eliminated

Author(s)	Year	Study design	Diagnosis	Patient number	Follow-up	Outcome
Kanpolat et al ²²	2009	Retrospective cohort	Cancer pain	193	Up to 6 mo	Significant reduction of VAS in > 90% of patients
Raslan ¹⁶	2008	Prospective open-label	Cancer pain	41	6 mo	Significant improvement in VAS, KPS, ADL, sleeping h at 6 mo
Raslan ²³	2005	Case series	Cancer pain	8	6 mo	Improvement of VAS, NRS, anterior transdiscal
Crul et al ²⁴	2005	Case series	Cancer pain	43	Up to 4 y	Significant and persistent reduction of NRS
Yegul and Erhan ²⁵	2003	Case series	Cancer pain	9	Short	Safe bilateral cordotomy after 1 wk
Jones et al ²⁶	2003	Case series	Cancer pain	9	Up to 830 d, median 107	Open cordotomy effective in pain reduction
McGirt et al ²⁷	2002	Case series	Cancer pain	6	5–11 mo, mean 6 mo	Effective significant reduction of VAS, using MRI-guided cordotomy
Jackson et al ¹⁰	1999	Case series	Cancer pain	52	1–52 wk	83% of patients had substantial pain relief initially
Sanders and Zuurmond ²⁸	1995	Case series	Cancer pain	80 (18 bilateral)	3–18 mo	87.1% with satisfactory pain relief initially, then gradually faded
Fenstermaker et al ²⁹	1995	Case series	Cancer pain	6	Short	Satisfactory pain relief in 5/6 patients; this is mainly a technical note
Nagaro et al ³⁰	1994	Case series	Cancer pain	10	94.7 d $\pm$ 71.1 d	Significant reduction of VAS from 8.5 to 3
Lahuerta et al ³¹	1994	Case series	Cancer pain	146	Up to 9 mo	Complete or satisfactory pain relief in 89% of patients
Krol and Arbit ³²	1993	Case series	Cancer pain	13	Short	Good pain relief in the short term

Stuart and Cramond ³³	1993	Large retrospective cohort	Cancer pain	273	Long term	Satisfactory pain relief in 89% of patients
Amano et al ³⁴	1991	Case series	Cancer pain	161 (mostly cancer)	> 6 mo	76% showed good pain relief
Höglberg et al ³⁵	1989	Case series	Cancer pain	24	> 6 mo	79% pain relief
Palma et al ³⁶	1988	Case series	?	?	?	?
Ischia et al ¹⁷	1985	Retrospective cohort of prospectively collected data (survival analysis)	Cancer pain	119	Until death (survival analysis)	92% initial pain relief declines to as low as 30% at the time of death (around 12 mo)
Ischia et al ¹⁸	1984	Retrospective cohort of prospectively collected data	Malignant vertebral pain	69	Median 5 mo	71% pain relief, some patients may be included elsewhere
Ischia et al ¹⁹	1984	Retrospective cohort of prospectively collected data	Cancer pain	36	Until death	Bilateral cases only, 47% complete pain relief and 12.5% pain relief; patients might be included in other studies
Cowie and Hitchcock ³⁷	1982	Case series	Cancer pain	33	Up to 1 y	95%, 73%, 55% pain relief at immediate postop, 6 mo, and 1 y, respectively
Meglio and Cioni ³⁸	1981	Case series	Cancer pain	52	15 wk	73% and 63% had excellent results after 1 and 15 wk, respectively
Lipton ³⁹	1978	Case series	Cancer pain	650	Until death	95% initial pain relief, drops to 75% near death; 6.2% mortality
Rothbard et al ⁴⁰	1972	Case series	Cancer pain (gynecologic)	10	Up to 3 y	Pain-free interval 6–29 mo

(Continued)

Table 23.1 (continued) Results of percutaneous cordotomy for cancer pain from 1966 to 2010 in all English-published peer-reviewed manuscripts, redundancy eliminated

Author(s)	Year	Study design	Diagnosis	Patient number	Follow-up	Outcome
Batzdorf and Weingarten ⁴¹	1970	Case series	Cancer pain	41/47 total	Up to 12 mo	27 excellent or good pain relief, 11 fair pain relief
O'Connell ⁴²	1969	Case series	Cancer pain (rectal)	56	6 wk–2 y	63% had complete pain relief until death
French ⁴³	1974	Case series	Cancer pain	177/200	Up to 1 y	90.6% had no pain or good pain relief, up to more than 1 y; open surgical cordotomy
Rosomoff ⁴⁴	1969	Case series	Cancer pain	71 cancer pain/ 100 total	Until death	Initial 93% pain relief that fades to about 60% at 2 y; this is a bilateral series
Lin et al ⁴⁵	1966	Case series	Cancer pain	38	2 wk–7 mo	74–86% adequate (no pain or good pain relief) depending whether it is anterior or posterior
Mullan et al ³	1965	Case series	Cancer pain	47	Short	36 good outcome, 1 death
Esposito et al ⁴⁶	1985	Case series	Cancer pain	8	6 mo	Cordotomy worked for 2–3 mo only
Grote et al ⁴⁷	1978	Case series	Cancer pain	138	Unclear	Initial relief in 114 (106 complete, 8 partial), dysesthesia in 9, 1 anesthesia dolorosa
Tasker ⁹	1976– 1977	Case series	Mostly cancer pain	264 mixed	Unclear	Initial 96%, 83% pain relief unilateral and bilateral; decreased to 89% and 66% at the time of last follow-up; 3% permanent complications
Crue and Felsoory ⁴⁸	1974	Case series	Mixed	29/48	Short	31% pain relief even in the short term
Fox ⁴⁹	1973	Case series	Mixed	14/18	Unclear	11/14 had adequate pain relief

Tasker and Organ ⁵⁰	1973	Case series	Mixed, mostly cancer pain	78 total	Until death, survival generally 3–6 mo	84% had pain relief till death
Smith ⁵¹	1972	Case series	Mixed, mostly cancer pain	19 total	Around 6 mo	In cancer pain patients, 100% complete or satisfactory pain relief
Foer ⁵²	1971	Case series	Cancer pain	21/30	Unclear	90% had partial to complete pain relief; 1 death
Salmon ⁵³	1969	Case series	Mixed	34 total	Unclear	25/34 had good pain relief, minor transient ataxia in 3, no mortality
Fox ⁵⁴	1969	Case series	Mixed	50	Not reported	Not reported
Uihlein et al ⁵⁵	1969	Case series	Cancer pain	45/50	Unclear	80% with either complete (52%) or satisfactory (28%) persistent pain relief
Acosta and Grossman ¹¹	1969	Case series	Cancer pain	12/15	3 mo–2 y	Adequate pain relief in all patients; 2 required redo
Kelly and Alexander ¹²	1966	Case series	Cancer pain	14/17	1–10 mo	13/14 had pain relief
Rosomoff et al ⁴	1966	Retrospective cohort	Cancer pain	82/100	Mostly 24 wk; by that time only 39/100 survived; the 61 deaths were cancer pain	Initial 92% pain relief, 82% at 24 wk
Rand et al ¹³	1965	Case report	Cancer pain	2	Short	One case had pain relief; it is a report about cooling electrode
Gildenberg ¹⁴	1976–1977	Retrospective cohort	Cancer pain	288	6 mo	82–85% had pain relief till 6 mo, depending on whether anterior or posterior
Raskind ¹⁵	1969	Case series	Mixed	30/237	Not stated	60% good pain relief, not requiring narcotics

Abbreviations: ADL, activities of daily living; KPS, Karnofsky performance scale; MRI, magnetic resonance imaging; NRS, numeric rating scale; VAS, visual analog scale.

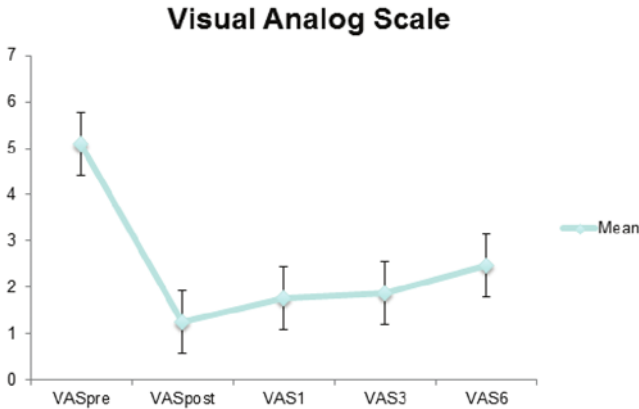


Fig. 23.3 Persistence of statistically and clinically significant reduction in visual analog scale after cordotomy at 6 months.

(a contraindication for cordotomy). True Ondine curse is universally fatal; however, no cases of Ondine curse have been reported so far using CT-guided cordotomy.^{16,22}

Postcordotomy dysesthesias affect 5% of patients and usually develop in long-term survivors who either have a significant component of neuropathic pain or had received a large lesion.^{56,57}

With direct visualization of the electrode in the spinal cord and proper electrophysiological verification, ipsilateral weakness should be a very rare occurrence. Ataxia and urinary incontinence have been reported by some authors, but not in modern cases of CT-guided cordotomy.^{22,56}

One rare, troubling, and unpredictable consequence of cordotomy is what is called mirror pain, which either can be attributed to a bilateral pain syndrome that is masked by marked severity on one side, or is potentially due to the bilateral projection of dorsal roots into both spinothalamic tracts.

Local anesthetic can be inadvertently injected into the CSF if mistaken for the water-soluble radiocontrast. This can lead to respiratory cessation, especially if a cisternal injection is performed. This underscores the need for clear marking of injection syringes and segregation of local anesthetic syringes from the surgical field.

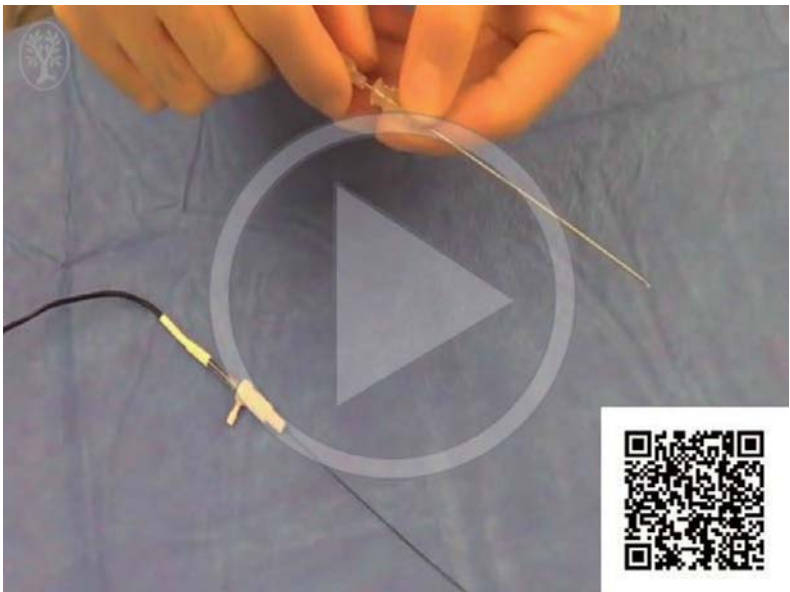
■ Bilateral Cordotomy

Bilateral cordotomy has been reported by both Kanpolat et al and Yegul and Erhan.^{22,25} It relies on a superselective lesion of the dorsal part of the spinothalamic tract close to the equator of the cord to avoid damage to the respiratory fibers that are in close proximity to the ventral component of the spinothalamic tract. The two sides should not be disrupted at the same session, and an interim interval of 2 weeks is recommended. Even with these restrictions, bilateral cordotomy can benefit bilateral lower extremity pain given the known somatotopic organization of the spinothalamic tract.

■ Future of Cordotomy

In medicine, historic procedures are sometimes reborn: What is old is new again. Cordotomy is one procedure that is poised to be utilized again for many reasons: (1) the increasing costs to maintain and manage the complications of intrathecal opioid systems; (2) the realization that pharmacological neuromodulation is not always reversible (e.g., weaning a patient from intrathecal opioids has in many instances proved impossible); (3) an increased recognition of opioid-induced hyperalgesia; (4) a need for the development of cost-effective procedures; and (5) the potential for increased safety, accuracy, and precision of a cordotomy procedure using technology such as intraoperative monitoring and neuronavigation.

Intraoperative imaging is making significant inroads in neurosurgery, and cordotomy may also benefit from this technology. A report of O-arm-guided (Medtronic, Minneapolis, MN, USA) cordotomy was recently published. The author has been using the intraoperative CereTom CT scanner (NeuroLogica, Danvers, MA, USA) for CT-guided tractotomy and myelotomy. With wider bore intra-operative CT scanners, cordotomy will move back to the surgical theater from the radiology suite. Endoscopic cordotomy has been introduced recently, and this technique presents the potential of improved visualization during the placement of the lesion electrode.⁵⁸



Video 4. Percutaneous CT-Guided C1-C2 Anterolateral Cordotomy

Editor's Comments

Cordotomy may be the exemplar for procedures that have proven effective in the past, were then lost to practice, and have been resurrected judiciously. Medicine, particularly pain medicine, is a trendy business. During the past several decades, neuromodulation has dominated the care of patients with chronic pain. When trainees never see a particular procedural option, those procedures tend to disappear; they are lost to future generations. This is not to say that we should uncritically adopt past practices, but more to reflect on what might be gleaned and improved on from older techniques.

CT-guided cordotomy applies established principles, and takes advantage of newer technology that can make the procedure safer and more effective. If cordotomy is to undergo a renaissance, it will be in the area of cancer-related pain. As I have pointed out in previous comments, the fact that at minimum 10% of cancer patients experience uncontrolled pain despite maximum tolerable medical therapy indicates that there is a role for surgical pain control in these individuals. Ablative procedures, when done properly by experienced clinicians, can provide effective pain control while preserving health care resources. As Drs. Raslan and Viswanathan mention, this conclusion is supported by evidence that the practice of cordotomy has been reinvigorated in countries that do not have the health care resources of the United States. As the proportion of our economy devoted to health care in the United States comes more into alignment with that of other countries, there will be more pressure to implement cost-effective pain-relieving procedures like cordotomy in patients with cancer-related pain.

As cordotomy re-emerges as a viable procedure, there will be a need to define its role, if any, in the treatment of pain not related to cancer. Several generations of neurosurgeons have passed since cordotomy was attempted for chronic "benign" pain. Careful and cautious inquiry may yield some instances when ablative lesions might produce durable pain relief.

Our first steps will be to reeducate a generation of neurosurgeons in the technique of cordotomy, probably CT-based. I am grateful to Drs. Raslan and Viswanathan for initiating that process.

■ Conclusion

Cordotomy is a very effective pain-relieving procedure that is most appropriate for patients with unilateral somatic cancer pain, who have a life expectancy of 1 to 2 years. It can be performed with the patient awake using CT guidance and myelography. It is a rather classic procedure that has a place in today's pain surgery practice.

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Midline Myelotomy and the Interruption of the Postsynaptic Dorsal Column Pain Pathway for the Treatment of Visceral Pain

Haring J. W. Nauta, Karin N. Westlund, and William D. Willis

This chapter attempts a synthesis of three topics: visceral pain, the surprising discovery of robust pain pathways in the dorsal columns of the spinal cord, and a body of knowledge around an old operation, midline myelotomy, now used only rarely. Each topic could provide material for a separate chapter, but they are discussed together here because the evolution of their understandings is closely intertwined so that each informs the other. Better understanding of the midline myelotomy operations used clinically and how they were effective¹ (see also **Fig. 24.1**) inspired laboratory studies in animals that led to a better understanding of the anatomy and physiology of the pain pathways that ascend in the dorsal columns. An improved understanding of visceral pain characteristics, in turn, and their differences from somatic pain (see **Table 24.1**) clarifies why visceral pain pathways and mechanisms might be different from those for somatic pain, and why in the past visceral pain seemed so difficult to treat. And finally, with an improved understanding about the postsynaptic dorsal column pathway and the properties of visceral pain, it has been possible to refocus the older myelotomy operations on the aspects that make them more effective and safer. The story has an almost circular quality of discovery and was laid out in great detail in the first edition of this text,² wherein the conclusions were still so novel that they were presented as tentative. Since then, further work in both the basic science laboratory and the clinical application has reinforced the conclusions. This is a process still in evolution and our increasing knowledge from the laboratory suggests that further clinical refinements may still be possible.

■ Clinical Implications

What we are successfully doing clinically today is treating intractable visceral pain of pelvic origin by interrupting an ascending postsynaptic pathway in the dorsal columns that conveys more visceral than somatic pain. For the pelvic and lower abdominal organs, the pathway runs in a juxta-midline direction, so the ascending axons there can be easily located and interrupted either by making a short transverse cut, or crush (see **Fig. 24.2a, b**), as we do it today in the punctate midline myelotomy (PMM) operation; or we can use “collateral damage” from a sagittal cut in the traditional midline myelotomy operation³ originally intended as a commissural myelotomy proposed by Greenfield in 1926. The PMM operation we perform today might more properly be described as a limited transverse dorsal column myelotomy or transection centered on the midline, to distinguish

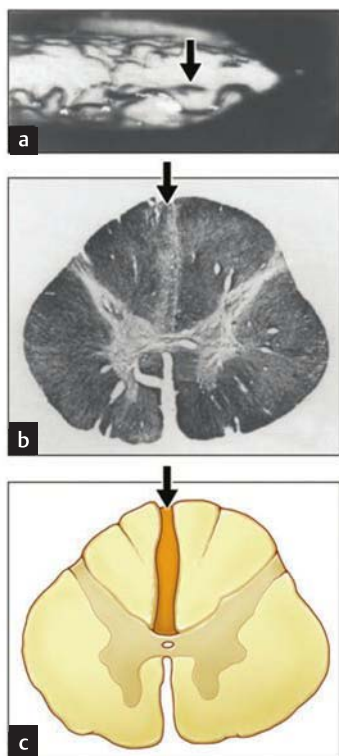


Fig. 24.1 Limited midline myelotomy as performed by Hirshberg with a short sagittal cut in the midline at the T10 level. **(a)** Intraoperative photograph. **(b, c)** The extent of the lesion when analyzed at autopsy (*arrows*). Note that although the myelotomy produced dramatic relief of chronic, medically intractable visceral pain, the extent of the lesion does *not* interrupt either the dorsal or ventral commissures. This observation was pivotal in inspiring further analysis into how the lesion could have been so effective when it clearly did not interrupt the bilaterally crossing fibers of the classically understood spinothalamic pathway. (Modified from Hirshberg et al.¹)

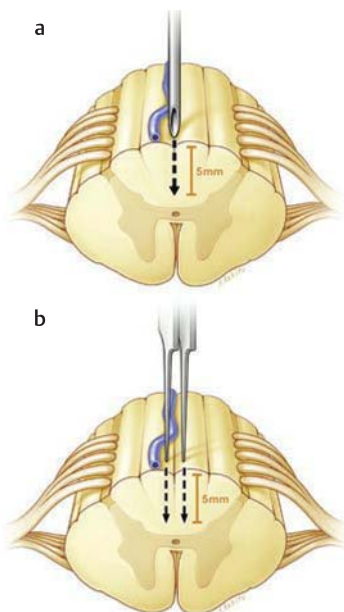


Fig. 24.2 **(a)** The transverse cut interrupting the medial millimeter of the dorsal columns on either side of the midline was originally made using the point of a 16-gauge needle as a knife. The needle tip was inserted to a depth of 5 mm precisely centered on the midline. No standard scalpel blade was narrow enough to produce the depth and width required. (Modified from Nauta et al.⁴¹) **(b)** The lesion was later made using a jeweler's forceps inserted as shown. (Modified from Nauta HJW, Hewitt E, Westlund KN, Willis WD. Punctate myelotomy for the relief of visceral cancer pain. *J Neurosurg Spine* 2000; 92:L25-L30.)

Table 24.1 Comparison of visceral and somatic pain

	Visceral pain	Somatic pain
Primary pathway	Postsynaptic dorsal column	Spinothalamic tract
Injury required?	No	Yes
Referral	Yes	No
Sensation	Continuous burning	Sharp
Localization	Poor	Good
Hyperalgesia	Yes	Yes
Adequate stimulus	Stretch, traction, inflammation, ischemia	Damage, infection
“Emotional valence”	Higher	Lower
Input to spinal cord	5–8%	95–98%
Central terminals	Highly arborized; end in laminae I, II, V, X, some on contralateral side	Discrete termination in superficial laminae
Somatotopic	No	Yes
Viscerosomatic convergence	Yes	Yes
Location in dorsal column	Midline dorsal column, intermediate septum	Majority of the dorsal column
Crossed pathway	No	Yes
First spinal cord relay	Neurons in laminae III–VII, X	Neurons in laminae I, IV, V
Second relay	Second dorsal column nuclei Third medial and intralaminar thalamic nuclei	Thalamus, ventral posterolateral (VPL) nucleus
Cortical termination	Limbic—cingulate, prefrontal, insular	Sensory neocortex—SI, SII cortex

it from the traditional midline myelotomy in which the cut in the spinal cord is in the sagittal plane. **Fig. 24.3** highlights this comparison.

■ The Anatomy and Physiology of the Postsynaptic Dorsal Column Visceral Pain Pathway

Fig. 24.4 (modified from Willis et al.⁴) summarizes what is now understood about the anatomy of the postsynaptic dorsal column pathways as these relate to visceral pain. The information comes from physiological localization studies as well as from retrograde and anterograde labeling studies in rats. **Table 24.2** highlights the differences between the long-recognized presynaptic dorsal column pathway and the more recently characterized postsynaptic dorsal column pathway.

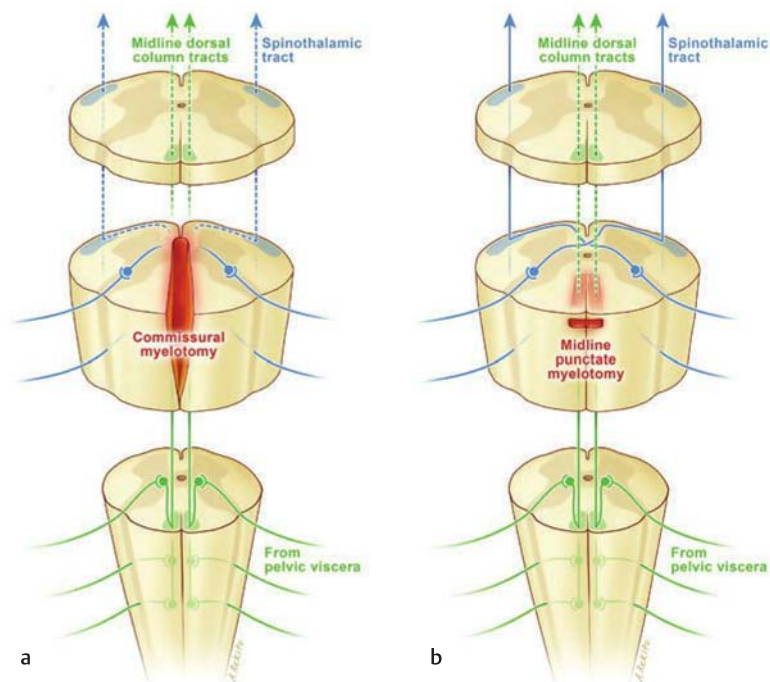


Fig. 24.3 Comparison of the effects of commissural myelotomy with punctate midline myelotomy (PMM). The commissural myelotomy (**a**) requires a sagittal cut deep enough to reach the commissures so that the bilaterally crossing fibers of the spinothalamic tracts will be interrupted. Somatic pain should thereby be affected over an area coextensive with the segmental levels involved. However, in doing so, the medial fibers of the dorsal columns will also be injured, interrupting the postsynaptic dorsal column pathway subserving predominantly visceral pain for all levels caudal to the lesion. In punctate midline myelotomy (**b**), only the postsynaptic dorsal column pathway is interrupted with a very short transverse incision centered on the midline, affecting mostly visceral pain caudal to the level of the lesion.

Postsynaptic dorsal column axons have their cell bodies in the gray matter of the spinal cord (see review by Willis and Coggeshall⁵). Many are concentrated in laminae III and IV of the dorsal horn, although they are found in other laminae as well.⁶⁻⁹

Retrograde labeling of axons in the dorsal columns of the cervical spinal cord has demonstrated the presence of a large population of postsynaptic dorsal column neurons in the area around the central canal of the sacral spinal cord (see **Fig. 24.5**).¹ It is known that visceral primary afferent fibers project to this region.¹⁰⁻¹⁴ Injection of an anterograde label into the same region of the spinal cord showed that neurons in this region project to the medial part of the gracile

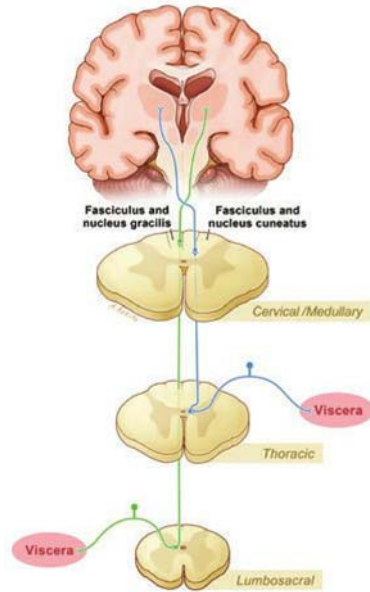


Fig. 24.4 Course of the axons of postsynaptic dorsal column neurons whose cell bodies are located in the central, visceral processing region of the spinal cord around the central canal. The projection from the sacral spinal levels ascends near the midline of the fasciculus gracilis. The projection from the midthoracic spinal cord levels ascends near the dorsal intermediate septum and ends in the lateral nucleus gracilis and medial nucleus cuneatus.

gray region of the sacral spinal cord (**Fig. 24.5**) that project to the medial nucleus gracilis.^{1,21,38} Anterograde tracing experiments using *Phaseolus vulgaris* leucoagglutinin (PHA-L)¹⁶ confirm that axons of postsynaptic dorsal horn neurons in the central gray region of the sacral spinal cord ascend in the fasciculus gracilis near the midline (**Fig. 24.4**). However, at least in rats, the axons of comparable neurons in the midthoracic spinal cord ascend more laterally, in the vicinity of the dorsal intermediate septum,^{15,16} which separates the fasciculus gracilis from the fasciculus cuneatus (also shown in **Fig. 24.4**). Many of the terminals of these axons are in the lateral gracile and medial cuneate nuclei, whereas others continue rostrally to higher brain areas.

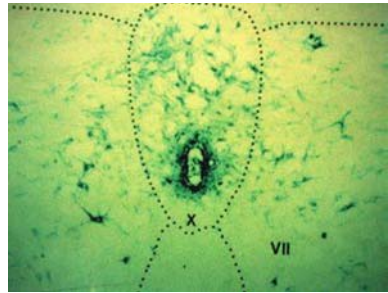


Fig. 24.5 Cells of the central, visceral processing region of the spinal cord around the central canal, retrogradely labeled by HRP injection into the dorsal column nuclei. These are the cells of origin of the postsynaptic dorsal column pathway. (From Hirshberg et al.¹)

nucleus.^{1,15,16} Thus, there is anatomical evidence of a postsynaptic dorsal column pathway that originates from a part of the spinal cord gray matter that is known to contain neurons that respond to somatic and/or visceral stimuli.^{17–20} The axons of these neurons in rats ascend in the fasciculus gracilis near the midline in a homologous position to the lesion made by Hirshberg,¹ as demonstrated after autopsy in his last clinical case (see **Fig. 24.1**).

Retrograde tracing experiments have confirmed the observation of a large population of postsynaptic dorsal column neurons in the central

Table 24.2 Comparison of presynaptic and postsynaptic dorsal column pathways

	Dorsal column pathways	
	Presynaptic: first order	Postsynaptic: second order
Nomenclature	Dorsal column pathway	Postsynaptic dorsal column pathway
Modality	“Epicritic,” vibration, discriminative touch, position sense, pressure	Visceral pain
Cells of origin	Dorsal root ganglia (DRG)	Spinal neurons in laminae III–VII, X Second order
Location in dorsal column	Majority of the dorsal column	Midline dorsal column–intermediate septum (fasciculus interfascicularis)
Myelinated	Yes	No
Termination site	Dorsal column nuclei	Dorsal column nuclei
Effect of midline lesion	Little or none	Reduces pelvic visceral pain

■ Evidence from Neurophysiological Studies in Animals

To determine if this postsynaptic dorsal column pathway carries visceral nociceptive information, it was necessary to demonstrate the response properties of neurons affected by this pathway. The first indication that cells present in the brain were visceral nociceptive neurons whose responses depended on information conveyed by the dorsal columns came from recordings of the activity of neurons in the ventral posterolateral (VPL) nucleus in rats.²² The neurons that were selected responded to noxious colorectal distension (CRD),²³ as well as to stimulation of a cutaneous receptive field. A lesion of the dorsal column at T10 was shown to dramatically reduce the responses of the VPL neurons to CRD and to weak mechanical stimulation of the skin, whereas a lesion of the lateral funiculus (and presumably of the spinothalamic tract) eliminated the responses to noxious mechanical stimulation of the skin (**Fig. 24.6**). By contrast, a lesion of the spinothalamic tract blocked the responses to noxious cutaneous stimulation but not those to CRD (**Fig. 24.7**). Comparable effects were also observed when acute inflammation of the colon by injection of a chemical irritant, mustard oil, was used instead of CRD to elicit a response. To ensure that it was the dorsal column projection that was involved in the responses of VPL neurons to CRD, a lesion study was done in which a radiofrequency or a kainic acid lesion was made *in* the nucleus gracilis.²⁴ A small, incomplete lesion of the nucleus gracilis resulted in a substantial reduction in the responses of VPL neurons to CRD, indicating that the pathway involved did relay in the nucleus gracilis.

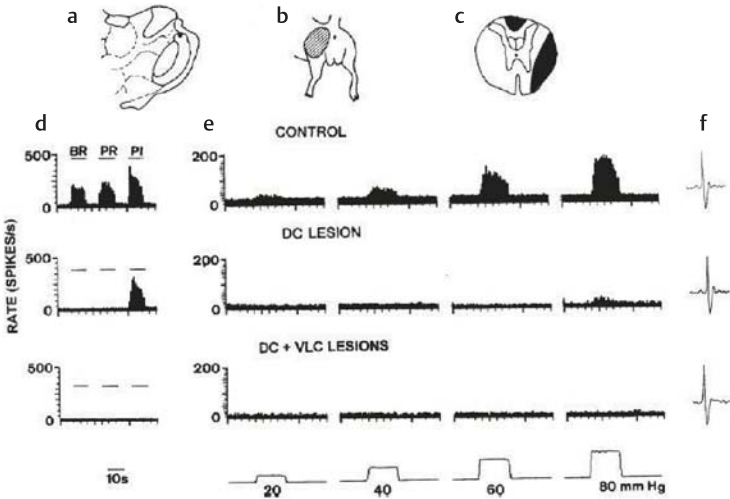


Fig. 24.6 Effects of spinal cord lesions on the responses of a neuron in the ventral posterolateral (VPL) thalamus of a rat to visceral and cutaneous stimulation. (a–c) The recording site, cutaneous receptive field, and locations of the lesions. (d) Responses to mechanical stimulation of the skin (brushing, BR, pressure, PR, and pinch, PI, due to application of arterial clips to a fold of skin). (e) Responses to graded intensities of colorectal distension (CRD), at bottom. The responses in the upper rows of (d) and (e) were recorded in the control condition; those in the middle rows followed a lesion of the dorsal column (DC); and those in the lower rows followed an additional lesion of the ventral lateral column (VLC). Note that the majority of the responses are eliminated by the dorsal column lesion, with the small remainder disappearing as well after the additional VLC lesion. The monitor traces below (e) show the pressures applied to the colon wall with intensities of 40 to 80 mm Hg considered noxious. (f) The spikes show that the recording conditions remained unchanged throughout the experiment. (Al-Chaer et al.²²)

Another question was whether the responses of VPL neurons to CRD depend on primary afferent axons in the dorsal column that project directly to the nucleus gracilis or on the axons of postsynaptic dorsal column neurons. The approach used to answer this question was to administer either morphine or the non-*N*-methyl-D-aspartate (NMDA) glutamate receptor antagonist 6-cyano-7-nitroquinoxaline-2,3-dione (CNQX) into the gray matter of the sacral spinal cord by microdialysis to interfere with synaptic excitation of postsynaptic dorsal column neurons following CRD²⁵ (Fig. 24.8). The responses of neurons in the nucleus gracilis to CRD were used to assay the effects of the drugs. Because drugs given by microdialysis are likely to affect only about one segment of the spinal cord,²⁶ the hypogastric nerves were sectioned prior to the experiment so that the responses of nucleus gracilis neurons to CRD depended on input to the spinal cord just through the pelvic nerves. The open dialysis membrane of the microdialysis fiber was placed in the gray matter of the sacral spinal cord. If the responses of neurons in the nucleus

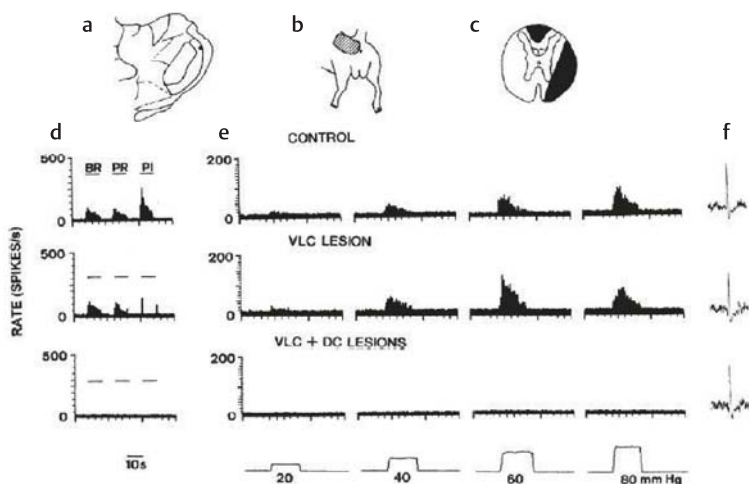


Fig. 24.7 Effects of spinal cord lesions on the responses of a VPL (ventral postero-lateral) neuron to cutaneous and visceral stimuli. The abbreviations and arrangement of data are the same as in **Fig. 24.6**. However, the sequence of the spinal cord lesions was reversed, so that the VLC (ventral lateral column) was interrupted first and then the dorsal column lesion. Note that only about a 5 to 10% reduction in the responses occurs after the VLC lesion whereas the responses are eliminated completely after the additional dorsal column lesion. Again, as shown in **(f)**, the recording conditions remained constant throughout the experiment. (From Al-Chaer et al.²²)

gracilis depended entirely on the activity carried there directly by primary afferent fibers, then morphine or CNQX administered by microdialysis into the sacral spinal cord gray matter would presumably not have any effect.^{27,28} However, administration of either morphine or CNQX did indeed block the responses of neurons in the nucleus gracilis to CRD, but not the responses to cutaneous input, which were presumably transmitted directly by ascending collaterals of primary afferent fibers. The action of morphine depended on activation of opiate receptors because this action was reversed by systemic administration of the opiate receptor antagonist naloxone. Thus, this study confirmed the hypothesis that visceral nociceptive information is relayed through postsynaptic dorsal column neurons in the spinal cord.

Recordings from postsynaptic dorsal column neurons in the central gray region of the sacral spinal cord confirmed that many of these cells could be excited by CRD and that their responses to CRD are blocked by microdialysis administration of morphine or CNQX. The axons of these postsynaptic dorsal column neurons could be followed by antidromic stimulation and mapping of the nucleus gracilis. Morphine administered locally into the sacral spinal cord by microdialysis showed a powerful effect on the responses of postsynaptic dorsal column neurons to noxious visceral stimuli. In contrast, morphine showed a weaker action on the responses of these same neurons to somatic stimuli. This differential action may

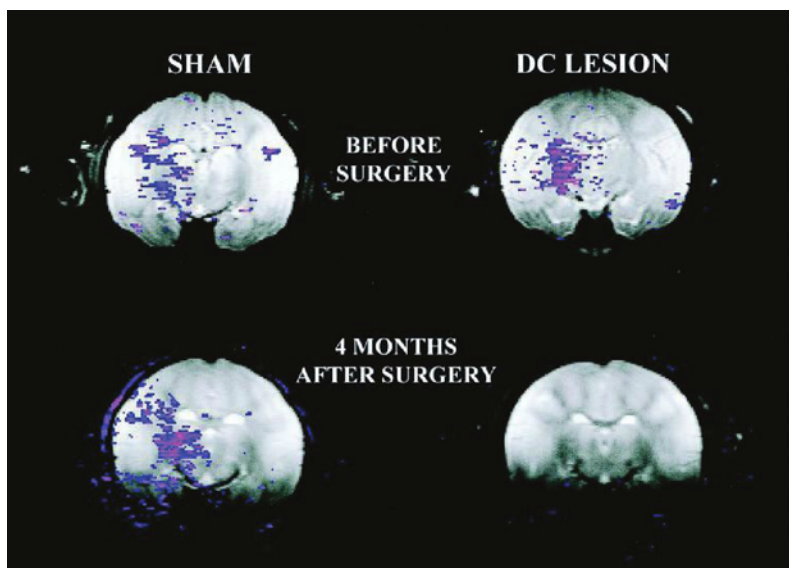


Fig. 24.8 Regional cerebral blood volume in response to noxious colorectal distension before and after a lesion of the dorsal column or sham surgery in anesthetized monkeys. The colored areas in the upper images represent voxels showing an enhanced regional blood volume in the brain of a monkey following noxious CRD (colorectal distension) as determined by functional magnetic resonance imaging (MRI). The transverse sections are taken through the posterior thalamus. The lower images were taken at approximately the same level and in the response to the same stimulus 4 months following sham surgery (*left*) or a lesion of the dorsal column (DC) at T8 (*right*). (Willis et al.³⁸)

be relevant to the effectiveness of epidural or intrathecal morphine administration in relieving visceral pain in many patients.

A related study was done in which distension of the duodenum in rats was employed rather than CRD.²⁹ Both behavioral responses (writhing responses) and electrophysiological responses of VPL neurons were employed to evaluate the effects of dorsal column lesions in their studies. A lesion placed at the midline of the dorsal column was without effect. However, lesions placed bilaterally at the position of the dorsal intermediate septum resulted in a dramatic reduction in writhing responses and in the excitation of VPL neurons in response to duodenal distension. The failure of a midline lesion and the success of more laterally placed lesions of the dorsal column are readily understandable based on the observation by Wang et al.¹⁶ that the axons of postsynaptic dorsal column neurons originating from the central gray region of the midthoracic spinal cord ascend near the dorsal intermediate septum, rather than near the midline (see **Fig. 24.4**). Lesions that have included this region have also been effective in restoring behavioral exploratory activity in a pancreatitis model in rats.³⁰ These findings, if applicable to humans,

suggest that surgical elimination of visceral pain that is relayed through the midthoracic spinal cord would require placement of lesions bilaterally near the border between the gracile and cuneate fasciculi. However, the situation in humans may be different because both Hwang et al.³¹ and Kim and Kwon³² report good results treating upper abdominal visceral cancer pain with PMM at upper thoracic levels, but the lateral extent of their lesions is not fully known.

Responses of neurons in the VPL nucleus in macaque monkeys can also be evoked by CRD.³³ A lesion of the dorsal column at T10 reduced the responses by about half. Additional lesions of the dorsal parts of the lateral funiculi caused a further reduction in the responses to CRD, but lesions of the ventrolateral white matter were not effective in changing the responses. The effectiveness of lesions in the dorsal part of the lateral funiculus in monkeys can be explained by the presence of axons of the lamina I component of the spinothalamic tract^{34,35} and some postsynaptic dorsal column neurons in this location.⁶ A comparable dorsal lateral funiculus projection of postsynaptic dorsal column neurons of the lumbosacral spinal cord does not occur in rats.⁸

A survey of postsynaptic dorsal column neurons and of spinothalamic tract neurons in the sacral spinal cord of monkeys revealed that the two types of neurons had similar stimulus-response functions following different intensities of CRD.³⁶ However, the sample included many more postsynaptic dorsal column neurons than STT cells. It was suggested that the greater effectiveness of the postsynaptic dorsal column pathway compared with the spinothalamic tract in conveying information to the brain about visceral pain could be based on the number of neurons available for activation by CRD in the two pathways.

Regional cerebral blood volume changes following CRD were mapped in the brains of four monkeys.^{37,38} A lesion was made in the dorsal column at T8 in three of the animals, and sham surgery was done on the others. Before surgery or after sham surgery, CRD distension produced increases in regional blood volume in many regions of the brain, including the lateral thalamus in the region of the VPL nucleus. Following the lesion of the dorsal column, CRD resulted in no regional blood volume changes (**Fig. 24.8**), suggesting that the lesion eliminated the neural connections necessary for activation of higher brain centers by nociceptors supplying the colon.

■ Multiple Pain Pathways: Some General Considerations

From the above review of anatomy and physiology, it seems clear that there are several pathways in the spinal cord white matter transmitting information about pain. This redundancy probably reflects at least two principles. First, visceral pain is different from somatic pain at the functional and perceptual levels (see **Table 24.1**), and it should therefore not be surprising that these two broad pain categories are mostly segregated into different anatomical systems. Second, redundancy and a degree of overlap between systems may reflect the importance of pain to survival generally from both a phylogenetic and an evolutionary perspective. Even nonmammalian vertebrates appear to have a postsynaptic dorsal column pathway in addition to the spinothalamic pathway as demonstrated in a reptile.³⁹

A closer look reveals several functional differences among the routes taken by these separate pain pathways. The spinothalamic tract pathway transmits precise body map information about pain arising from somatic structures (i.e., skin, muscles, bones) to higher brain centers. Many spinothalamic tract neurons are located in deep laminae IV, V, and VII of the dorsal horn and send their axons through the well-known ventral or “anterolateral” white matter pathway. The integrated information carried can arise from both somatic and visceral structures, and is likely responsible for transmission of referred pain and chronic pain states. Information about temperature and acute somatic pain is primarily carried by axons located in the lateral white matter from spinothalamic tract neurons in the superficial lamina I.^{35,40} However, pain arising from visceral structures is relayed in large part by the dorsal horn postsynaptic dorsal column cells in lamina III and situated medially in laminae IV–VII and X. The visceral pain information is transmitted by way of the dorsal column–medial lemniscus pathway. Despite this association with the dorsal column–medial lemniscus pathway carrying discriminative sensory information, visceral pain is diffuse and poorly localized. This is probably explained by the widely divergent extent of the visceral afferent terminal endings that can communicate with numerous spinal cord neurons. In comparison, the terminal endings in the superficial spinal cord convey the discrete, somatotopic information in the spinothalamic tract that allows the precise body map localization of painful input through the lateral thalamus to the sensory cortex. The visceral pain information is also transmitted to the medial and intralaminar nuclei of the thalamus. Thus, the visceral sensory and pain pathways are biased toward limbic cortical areas, where they are potentially better able to influence behavioral and emotional response mechanisms.

■ Initial Clinical Application with the Intention to Interrupt an Ascending Visceral Pain Pathway by Making a Short Transverse Cut across the Midline of the Dorsal Columns

In the late 1990s our group began a prospective study to determine the usefulness of making a small *transverse* cut in the dorsal columns for the treatment of intractable pelvic visceral pain. This represented a significant departure from the usual midline myelotomy procedure, where the incision in the spinal cord had always been made in the midsagittal plane, and any damage to the dorsal columns had been unintentional but fortuitous. We undertook this departure because: (1) convincing laboratory evidence had accumulated demonstrating that there is an important visceral pain pathway in the dorsal columns; (2) for the pelvic visceral organs this pathway runs in an identifiable place along the dorsal midline of the spinal cord; (3) there is historical clinical evidence in humans that limited dorsal column lesions do not produce disabling deficits; and (4) many past published clinical observations about midline myelotomy were consistent with the conclusion that functional deficits were minimal and that the benefits in visceral pain control were substantial.

The first case⁴¹ was a 39-year-old diabetic woman who had been successfully treated for cancer of the uterine cervix by radiation. However, although her tumor

was well controlled, she had severe pain apparently due to radiation damage of the bowel, bladder, and ureter. The pain was not controlled by high doses of opiate analgesics, and she had excruciating pain during bowel movements. Her weight had decreased from 96 to 75 lbs, a life-threatening problem. She described herself as either in severe pain or drowsy from overmedication with less severe pain. She had withdrawn socially, and saw no end in sight since her tumor appeared to be well controlled. She requested surgery after full disclosure and institutional review board (IRB) and ethics committee criteria were met. At operation, after T8 laminectomy, a small transverse cut was made across the midline of the dorsal columns ending 1 mm to each side of the midline. The transverse cut was so short an ordinary scalpel blade was too large to allow it. Instead, we made it by using the sharp edge of a 16-gauge needle as a microknife, inserting it vertically into the midline of the spinal cord to a depth of 5 mm as shown in **Fig. 24.2a, b**. Also, for this reason we initially called the procedure “punctate midline myelotomy (PMM),” but it differs from past midline myelotomy procedures because it is made in the transverse rather than in a sagittal direction. When performing the operation with such a narrow cut, we considered it extremely important that the patient and microscope be positioned so that the midline view is perfectly vertical (see **Fig. 24.9a**) and that the cut or crush not deviate from the midline at the depth. We also observed that the usually midline dorsal vein may meander quite extensively off midline (see **Fig. 24.9b**), and the guide to true midline should be determined by the shallow sulcus seen in a position midway between the dorsal root entry zone on each side.

Immediately postoperatively the pain that led to the operation was relieved completely. As expected, the patient’s operative-site pain was initially difficult to control, consistent with her chronic opiate usage leading to downregulated opiate receptors. However, within a few days the patient’s narcotic medication could be reduced dramatically, and she soon thereafter regained her appetite and part of her lost weight. She resumed social interactions. About a year later, she developed a fistula between the rectum and bladder and then another fistula between the bladder and peritoneal cavity. To treat these complications of her radiation, a colostomy was required as well as later insertion of nephrostomy tubes bilaterally. She had postoperative pain of the abdominal wall at the colostomy site that was managed by an increase in opiate medication. She also later experienced pain in her upper abdomen from peritonitis, but none below the umbilicus. Pain from the nephrostomies and sacral pain that developed later from a decubitus ulcer also could be managed with opiates. She eventually died 31 months after the punctate midline dorsal column myelotomy. During the postsurgical survival period, she never again experienced the severe pelvic pain refractory to high-dose opiates that led to the midline myelotomy.

Following that first case, we reported on PMMs performed on an additional five patients.⁴² The procedure was modified by using microforceps for blunt crushing of the juxta-midline dorsal column tissue to 1 mm on each side of the midline to a depth of 5 mm (see **Fig. 24.2b**). The modification was designed to minimize the chances that the procedure would result in bleeding within the spinal cord.

The procedure resulted in a significant reduction in patient ratings of “worst pain” and in daily narcotic use. Postoperative pain from the laminectomy, as well as opiate withdrawal symptoms, were transient and managed with gradual withdrawal of opiates. All but one of the patients became at least temporarily



Fig. 24.9 (a) The operative alignment of the microscope should be as close to the patient's vertical midline as possible to optimize the narrow vertical lesion along the midline of the dorsal columns. Every effort should be made to lesion only the tissue of the exact midline extending 1 mm to either side. (b) An example of a meandering midline dorsal vein, emphasizing that the localization of the midline should be based primarily not on the location of the vein, but on the midline contour of the dorsal columns, measured halfway between the dorsal root entry zone on either side.

independent of narcotic analgesics before other complications of the underlying disease intervened. There was no evidence of sensory deficits, motor weakness, gait disturbance, or sphincter dysfunction, although one patient had transient bilateral tingling in her toes and transient incomplete bladder emptying. Four of the patients died of tumor progression after survival times of 3 to 9 months. The remaining patient enjoyed 18 months of dramatic relief of pain due to a presacral lesion treated with radiation after biopsy. The tumor then progressed with spread into the sacrum, leading to the recurrence of pain attributable to compression of the sacral plexus and nerve roots within the sacral spinal canal. An aggressive resection of the tumor was then carried out with a combined anterior and posterior approach that included colostomy and resection of the sacrum from S3–S5 and

of the coccyx, as well as an epidural dissection of the caudal nerve roots. Three months after the resection, the pain was diminished and narcotic analgesics again could be discontinued.

The results of “punctate midline myelotomy” in our series of six patients indicate that this procedure can result in a significant reduction in visceral pain and in the requirements for narcotic analgesics in patients with pelvic and abdominal cancer. The benefits are uncertain for components of pain of somatic origin. Placement of the lesion transversely across the midline of the posterior columns 1 mm to each side would be expected to interrupt axons carrying pain signals from visceral organs that are relayed in the sacral spinal cord.^{1,16,22,25} However, we initially expected that more lateral lesions would probably be required to interrupt axons carrying pain signals from visceral organs that have primary afferent fibers that relay in the midthoracic spinal cord.^{16,29} Soon after these observations, Kim and Kwon³² reported relief of pain from stomach cancer by PMM at a high thoracic level. Hwang et al.³¹ also reported success with PMM at the T3 level to treat intractable visceral cancer pain of hepatobiliary or pancreatic origin.

The visceral pain that led to the midline myelotomy did not recur within the 3- to 31-month survival periods of this series of patients, although pain from other sources did develop in some of the patients. There were no long-lasting sensory, motor, or autonomic changes as a result of the procedure, although one patient had transient sensory changes and urinary retention.

Since the original case was published by Nauta et al.,⁴¹ several other series have reported good results with minimal or no deficits with the PMM procedure.^{31,32,43–45} In addition, an excellent review article has appeared by Hong and Andréén-Sandberg.⁴⁶ The reports of Kim and Kwon³² and Hwang et al.³¹ are particularly interesting because they performed the myelotomy at high thoracic levels for the treatment of pain derived from upper abdominal viscera and still were successful despite evidence in rats that axons of the postsynaptic dorsal column pathway for these organs might begin to ascend in a more lateral position in the dorsal columns.^{29,30}

■ Is It Safe to Transect Part of the Dorsal Columns?

The sensory modalities in humans that depend on information ascending in the dorsal columns are said to include discriminative touch, vibration sense, and proprioception.^{47–49} The idea of intentionally transecting part of the dorsal columns was, and remains, a strategy accepted only with great caution. This issue needs to be addressed with some emphasis because many clinicians are unlikely to accept an ablative procedure of even a small part of the dorsal columns without considerable reassurance. The evidence comes from both clinical observations in humans and experimental studies in animals that even large incomplete lesions of the dorsal columns do not appear to produce major disabling deficits. Qi et al.,⁵⁰ from experiments in squirrel monkeys, point out that part of this phenomenon may come from reorganization in the somatosensory cortex, with reactivation based on remaining secondary afferents from other sensory pathways originating in the spinal cord. McKenna and Whishaw⁵¹ report that in the rat, in the absence of dorsal columns, other sensory-motor pathways support a surprising degree of preserved function. Especially pertinent to this, in humans, Cook and Browder⁵²

performed dorsal column cordotomy at a midthoracic level in five patients and in the upper cervical area in three patients. They concluded that deficits were transient and did not include the expected major loss in proprioception. Noordeno-bos and Wall⁵³ also noted a remarkable degree of preservation of diverse sensory functions, including proprioception, in a clinical case of spinal cord transection with complete transection of both posterior columns and preservation of only part of one anterolateral quadrant.

Certainly any neuroablative procedure prompts concern about loss of function, but for small lesions of the dorsal columns, with preservation of other sensory systems, the deficits are not likely to be disabling, if they are detectable at all. This realization permits us to balance the benefits of pain control from a very limited transection of the dorsal columns against drawbacks that include minor demonstrable deficits, if any occur at all. This comes as something of a surprise to the neuroscience community because dorsal column lesions have long been equated with the disabling proprioceptive loss once seen commonly in *tabes dorsalis*. But that disorder is more properly considered to result from a loss of dorsal root ganglia neurons all up and down the spinal cord bilaterally. Witness the profound loss of deep tendon reflexes seen in *tabes dorsalis* as evidence of a more widespread sensory loss than would be suggested by the focal dorsal column loss seen in the Weil–Weigert stained histology of the postmortem spinal cord. Because most axons in the dorsal columns are presynaptic, with cell bodies of origin in the dorsal root ganglia, the dorsal columns will show significant axonal loss in the case of advanced *tabes dorsalis*, whereas the major deficits probably come mostly from other connections of the missing dorsal root ganglion neurons that function also at segmental and propriospinal levels, and transsynaptically through other ascending pathways such as the spinocerebellar tracts and the anterolateral quadrant system. Further reassuring is the general agreement that, unlike the anterolateral quadrant pathways, there are no major commingled descending motor pathways in the dorsal columns, and effects on respiration and sphincter control are not reported as a problem with dorsal column lesions.

■ How Historical Midline Myelotomy Operations Suggested the Presence of Pain Pathways in the Dorsal Columns

For unilateral somatic cancer pain, the development of anterolateral cordotomy was a logical choice. The procedures could be performed minimally invasively at the C1–C2 level with fluoroscopic guidance and physiological confirmation, and the clinical results matched the understanding of the anterolateral-quadrant spinothalamic pathway's role in the conduction of pain from the body wall and extremities. However, for midline or visceral abdominal pain, the anterolateral cordotomy was difficult to apply for two reasons. First, bilateral lesions would be required, and these resulted in unacceptable complications stemming from collateral damage to commingled descending pathways in the anterolateral quadrant related to respiration (“Ondine curse”) and loss of sphincter control. Second, even when efforts were made to stagger the lesions at different segmental levels, the results of mid-

line or visceral pain control were often disappointing. In retrospect, we can now understand these observations because for visceral pain, the dominant ascending pathway is the postsynaptic dorsal column system (see **Figs. 24.4, 24.6, 24.7**).^{1,21,22}

In 1926 the neuropathologist G. Greenfield proposed a solution³ to the problem of side effects from bilateral anterolateral cordotomies: interruption of the crossing axons of spinothalamic tract neurons on both sides by a longitudinal incision placed along the midline of the spinal cord. This procedure, originally intended to be a commissural myelotomy, was first performed by Armour³ in 1926 (reported in 1927) and later by many others. The success rate for the relief of cancer pain was relatively good, although reports of side effects limited enthusiasm for the procedure. If one performs the procedure with the goal of commissural myelotomy, then the first obstacle to clinical application is determining the functional segmental levels involved, something that might be difficult to discern from the description of a patient distraught with pain. Next, a multilevel laminectomy would be required and a similarly long sagittal incision made in the spinal cord, something that could easily result in vascular damage to the cord. Even with modern surgical instrumentation and techniques there is still a significant incidence of new postoperative deficit.⁵⁴ Although the results for visceral pain relief remain good, and if there is coexisting somatic pain, the commissural myelotomy procedure might still hold some advantages in affecting both anterolateral quadrant somatic and dorsal column visceral pain pathways.

The demonstration by Hirshberg et al.¹ that midline myelotomy could be dramatically successful without any damage to the commissures (see **Fig. 24.1**) certainly inspired renewed interest by laboratory researchers receptive to the idea that a pain pathway exists in the dorsal columns.

Seen now in retrospect, many reports by different authors can be reinterpreted in light of the knowledge that there is a pain pathway predominantly carrying visceral pain ascending near the midline of the dorsal columns. Many clinicians have reported successful pain control with operations on the spinal cord that traversed the dorsal midline in order to reach some intended deeper target. They attributed the successful pain control to the destruction of the target at that depth and made every effort to avoid damage to the dorsal columns in reaching this target. They worked with the understanding that the dorsal columns conveyed only “epicritic” sensory modalities and that any damage to them should be minimized to the extent possible. Furthermore, they reasoned that damage to the dorsal columns could not be expected to contribute to pain control because at that time they were not aware of any information about pain pathways ascending in the dorsal columns or their location specifically in the midline area they were traversing. Thus, they failed to appreciate that the even minimal damage they produced to the midline of the dorsal columns was possibly the dominant factor in determining the operation's success. Hitchcock⁵⁵ made a percutaneous lesion traversing the posterior midline at the C1 level and produced the intended pain relief in the bilateral upper extremities as well as an unexpected loss of pin prick pain in both lower extremities. He eventually seemed to prefer the explanation that stereotactic myelotomy at C1 interrupted a “central multisynaptic pain pathway” separate from the spinothalamic tract. Schvarcz^{56–58} also made lesions at the midline in the C1 segment to treat pain. Stimulation before lesion production caused sensations in the legs or over wider areas of the body and even the face. In addition to paresthesias, stimulation sometimes caused “bilateral burning truncal sensations.”^{56,57}

The lesions were made at a depth of 5 mm below the posterior surface of the spinal cord. Neuropathic pain (causalgia, postherpetic neuralgia, brachial plexus avulsion, and spinal cord lesions) was relieved in 64% of 14 patients who were followed for 0.5 to 4 years. No neurologic side effects of the surgery were seen. In a later report, the results in 79 cases of patients with intractable pelvic cancer pain who underwent the procedure were described. Satisfactory pain relief was obtained in 76% when defined as no pain or infrequent pain relieved by nonnarcotic analgesics. The patients were followed for 0.5 to 30 months, although most died of their malignant disease in the first 6 months.

■ Clinical Indications for PMM

We now appreciate that punctate or other limited posterior column myelotomies interrupt an ascending pain tract largely subserving visceral pain. There have already been several additional series reported from around the world, but it remains to be seen whether this operation will actually gain wider clinical acceptance and use. The six patients in our original series took over 2 years to acquire. At least in part because of our scientific interests, we limited our indications narrowly to patients with pure or predominantly pelvic visceral pain (as best as we could determine). However, when the visceral pain component is the most troubling to the patient, and its severity appears to be the source of inadequate response to opiates, it makes sense to treat the patient with midline myelotomy even if other somatic pain components are present because these likely could be managed by conventional medical means postoperatively. Such patients might remain dependent on opiates because of their residual somatic pain and might therefore be classified as treatment failures of midline myelotomy, but their overall status would likely be improved. By this reasoning, the indications for midline myelotomy might legitimately be broader than those used in our originally focused study. Hwang³¹ and Kim and Kwon³² have also demonstrated that it may be reasonable to broaden the indications for PMM to include patients with cancer pain from upper abdominal organs, including those of gastric, hepatobiliary, and pancreatic origin.

Patients suffering from cancer typically have already had surgery of one sort or another before being considered for myelotomy, and they typically are reluctant to undergo further open surgery for pain if there is any reasonable alternative. To address this limitation, there is room for future refinements of the posterior column myelotomy procedure, and following Hitchcock's⁵⁵ and Schvarcz's⁵⁶⁻⁵⁸ examples, a percutaneous method at the C1 level may be reasonable, especially now that advances in medical imaging are finding more widespread use in guiding therapeutic interventions. Toward this goal, Vilela Filho et al.⁴⁴ have already described successful application of a computed tomography (CT)-guided percutaneous technique for PMM in two patients. Also, CT-guided percutaneous techniques for extralemniscal myelotomy, as described by Kanpolat,⁵⁹ may have direct applicability with only minor modification to PMM. A percutaneous method at a thoracic level, however, may be problematic because at thoracic levels the interlaminar space is typically small (making needle entry difficult), whereas the septum posticum of the arachnoid and its associated midline dorsal vein is typically well developed,⁶⁰ tending to divert a needle off the targeted midline. Regardless of the rostrocaudal level, there

may be a hazard of subarachnoid hemorrhage with the percutaneous techniques using a 16-gauge needle as originally described,^{41,42} and it may be appropriate to transition to a fine-tip probe with less chance of vascular injury. However, even if a convenient percutaneous method for PMM could be developed, the bias against surgical ablative interventions for pain may persist. For example, percutaneous C1–C2 anterolateral cordotomy is not commonly performed today, although the technique has been well worked out for decades, it is minimally invasive, and the scientific rationale seems well established. The oral or transdermal medical alternatives or opiate infusion remains more attractive for many patients who are well controlled and who do not envision a long survival with the serious side effects of chronic opiates. For these reasons, it remains likely that the PMM procedure will find its use limited to exceptional circumstances, such as a patient with predominantly visceral pain refractory to opiate management, who is otherwise in good enough medical condition, and who has good enough tumor control that the side effects of chronic high-dose opiate therapy are anticipated to be a serious long-term problem—including drowsiness, which frustrates the enjoyment of any remission.

In some settings, the advantages of PMM over high-dose medication may also include cost concerns and independence from medical attention required with long-term high-dose oral, transdermal, or neuraxis infusion for opiate therapy.

■ Conclusion

There appears to be a clinically significant pain pathway that ascends in the dorsal columns. The pathway appears to subserve mostly visceral pain with a relatively minor role in somatic pain. Punctate midline myelotomy (PMM) interrupts components of this pathway related to the pelvis and lower abdomen and has also been modified successfully to treat visceral pain derived from upper abdominal

Editor's Comments

Dr. Nauta and his colleagues have developed a procedure based on a new anatomical substrate, the postsynaptic dorsal column pain pathway. In many ways, this represents a model of how new surgical procedures should be developed. The clues to its potential efficacy came from the analysis of the results of more historic procedures, such as midline myelotomy and midline C1 lesions. The implications of these older results were then taken as impetus to unravel the neuroanatomy of the dorsal columns, resulting in the discovery of a previously unrecognized tract in the medial ventral dorsal column. From this discovery, a procedure was developed to disrupt this tract in patients with pelvic visceral pain. In this case, the “bench” informed the “bedside”—a surgical approach based on solid basic research. Would that all surgical procedures had this pedigree!

The potential generalizability of their results will depend on data from other practitioners taking notice of this, and similar, procedures and publishing results. This will also require a change on the part of oncologists, who have proven very difficult to convince that surgery for pain secondary to malignancy is *ever* indicated. Clearly, as Nauta and colleagues point out, minimally invasive adaptations of the punctate myelotomy must be developed, if this procedure is to become more than a footnote in the surgical management of pain. It is my hope that reiteration of their approach in this book will both perpetuate these insights and promote the appropriate expansion of this procedure for the benefit of patients with intractable pain.

viscera. PMM can be useful for reducing otherwise intractable visceral pain due to cancers of the pelvis and abdomen in cases where these are not relieved by opiate analgesic drugs or where prolonged opiate therapy with major side effects is anticipated. The relief from posterior column “punctate” midline myelotomy has been observed to last for periods of up to 31 months after surgery without sensory, motor, or autonomic complications, and with no proprioceptive dysfunction. A growing appreciation of the anatomy, physiology, pharmacology, and results of clinical manipulations of the dorsal column visceral pain pathway may continue to contribute to better methods for pain control. It may also be possible to adapt PMM to CT or other image-guided percutaneous methods that would increase its applicability and attractiveness in more clinical settings.

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Dorsal Root Entry Zone Lesions

Marc Sindou

In the 1960s the “gate control” theory drew the attention of neurosurgeons to the dorsal horn as the first important level of pain modulation. This theory also suggested that it could be a target for pain surgery.¹ It gave rise to a popular method of electrostimulation of the primary afferents to the spinal cord, the spinal cord stimulation (SCS),² and also to destructive surgery in the dorsal root entry zone (DREZ) for very selected cases.³

The DREZ region was defined as an entity including the central portion of the dorsal rootlet, the medial part of the tract of Lissauer (TL), and layers I to V of the dorsal horn (DH), where the afferent fibers terminate and synapse^{3,4} (**Fig. 25.1**).

The first attempts of DREZ lesioning, using microsurgical coagulations, were performed at the University Neurological Hospital in Lyon, France, first for pain due to thoracic apex tumors (March 1972), then for neuropathic pain after spinal cord injury (December 1972), postamputation pain (July 1973), and brachial plexus avulsion pain (January 1974). Soon after, in September 1974, Blaine Nashold at Duke University, Durham, NC, started to develop DREZ lesioning using radiofrequency (RF) thermocoagulation,^{5,6} especially for pain related to brachial plexus avulsion. Other lesion makers were also used: the laser by Levy et al⁷ and Powers et al,⁸ and the ultrasound probe by Kandel et al⁹ and Dreval,¹⁰ again for pain caused by plexus brachial avulsion. More recently, based on the greater softness of the dorsal horn bordered by the tougher columns of the white matter tracts, Spaic et al proposed the aspiration of the gray matter over several metameres of the spinal cord through a single-level laminectomy.^{11,12}

■ Indications

The main indications for DREZ surgery correspond to clear-cut etiologies and mechanisms. Pain after brachial plexus avulsion is the most appropriate indication because it is predominantly linked with a deafferentation mechanism, and the same applies for pain after lumbar–sacral plexus injury. A similarly appropriate indication is pain after spinal cord injury, especially the one located at the conus medullaris/cauda equina, when pain is predominantly “segmental,” that is, corresponding to the segments injured. Pain after peripheral nerve lesions, amputation, or herpes zoster may be considered only if the predominant components of pain are of the paroxysmal and/or allodynic types, and only if spinal cord stimulation tried as the first option has failed. The same applies for complex regional pain syndrome (CRPS). Pain related to a malignancy may also be an indication, but only if of limited extent and in patients with a life expectancy measured in years. Separately, pain linked to intense spasticity in severely disabled patients can also be considered for DREZ surgery.

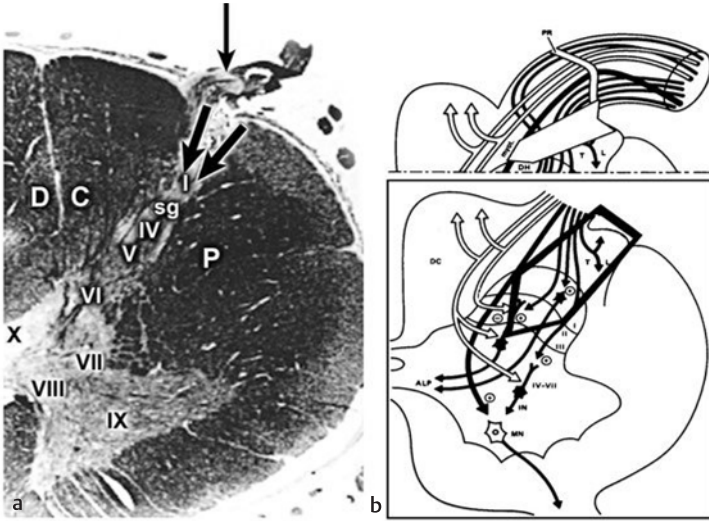


Fig. 25.1 Anatomical organization of the dorsal root entry zone (DREZ). As shown in the upper right diagram, each rootlet is divided owing to the transition of its glial support into a peripheral and a central segment. The transitional zone between the two segments is at the pial ring (PR). (a) Transverse hemisection of the spinal cord (at lower cervical level) with myelin stained by luxol-fushine, showing dorsal horn (DH) Rexed lamination (I–VI). Thinner arrow designates the transitional zone. (b) Organization of fibers (arrowheads). The micro-surgical DREZotomy (MDT) target includes the lateral bundle of the fine (nociceptive) fibers, the medial (excitatory) part of the tract of Lissauer (TL), the five dorsal-most layers of the DH where the primary afferents terminate and whose neurons become hyperactive if deafferented. MDT attempts to spare the maximum of the large (likely inhibitory) fibers of the medial bundle that reach the dorsal column (DC). P, pyramidal tract; sg, substantia gelatinosa.

■ Principles of Microsurgical DREZotomy

My preference is to perform DREZ surgery using the microsurgical techniques, since lesioning can be done relatively safely under direct vision after opening the dorsolateral sulcus.

Working with DREZ requires knowledge of the microsurgical anatomy of the spinal roots and cord,^{13,14} as well as of the internal morphology of the cord, to avoid damaging neighboring anatomical structures (**Fig. 25.2**).

Microsurgical DREZotomy (MDT) consists of a longitudinal opening of the dorsolateral sulcus performed ventrolaterally at the entrance of the rootlets into the sulcus, then of continuous microbipolar coagulations inside the sulcus down to the dorsal horn, along all the selected spinal cord segments to be lesioned. The lesion penetrates the lateral part of the DREZ and the medial part of the tract of Lissauer, and extends to the dorsal horn, which can be recognized by its pink-brown-gray color. Lesions are 2 to 3 mm deep, oriented medially and ventrally in the axis of the gray matter (**Fig. 25.3**).

Fig. 25.1 Considerations

Each dorsal root divides into 4 to 10 rootlets according to metameres.^{13,14} Each rootlet, of 0.25 to 1.50 mm in diameter according to levels, can be considered an anatomical–functional entity, that is a root in miniature.^{3,4} Each rootlet is divided owing to the transition of its glial support into a peripheral and a central segment. The transitional zone between the two segments is at the pial ring (PR), which is located approximately 1 mm outside the penetration of the rootlet into the dorsolateral sulcus. Peripherally, the fibers are mixed together. As they approach the PR, the fine fibers (considered nociceptive) move toward the rootlet surfaces. In the central segment, there is a spatial segregation of the afferent fibers according to their size and destination, with the fine fibers regrouping in the lateral region of the DREZ (dorsal root entry zone) to enter the dorsal horn (DH) through the tract of Lissauer (TL), and the large fibers in its medial region to reach the dorsal column (DC). The large myotatic fibers (myot.) are situated in the middle of the DREZ to project onto the motoneurons (MN) in the ventral horn. The DH is segmented according to Rexed lamination (I–VI). The TL is situated dorsolaterally to the DH apex, and includes two parts. (1) Its medial part—which the small afferents enter and where they trifurcate to reach the DH, either directly or through a few metameres ascending or descending pathway—transmits the excitatory effects of each dorsal root to the adjacent metameres.^{52,53} (2) Its lateral part—through which a large number of longitudinal endogenous proprio-spinal fibers interconnect different levels of the substantia gelatinosa (sg)—conveys the inhibitory influences of the sg to the neighboring metameres.⁵³ Most of the fine nociceptive afferents, which convey excitatory input, enter the DH through the TL medial part and the dorsal aspect of the sg. The Ramon y Cajal's recurrent collaterals of the large primary afferent fibers⁵⁴ approach the DH through the ventromedial aspect of the sg to exert inhibitory effects on the DH neurons.⁵⁵ Because a number of dendrites of the cells of origin of the spino-reticulo-thalamic tract, that will form the contralateral anterolateral pathways (ALP), make synaptic connections with the primary afferents inside the sg layers, the sg exerts a strong segmental modulating effect on the nociceptive input.⁵⁶

The procedure is intended to preferentially interrupt the (nociceptive) fibers grouped in the lateral bundle of the dorsal rootlet and the (excitatory) medial part of the tract of Lissauer. The dorsal-most layers of the dorsal horn, which harbor hyperactive neurons in the cases with deafferentation^{15–18} (**Fig. 25.4**), are destroyed if microcoagulations are performed deep inside the dorsal horn. The procedure is presumed to partially preserve the (inhibitory) medially located structures of the DREZ, namely the fibers reaching the dorsal column and their recurrent collaterals to the dorsal horn. MDT was conceived to avoid total abolition of tactile and proprioceptive sensations when preoperatively present, as well as further deafferentation.¹⁹ The depth and extent of the lesion are tailored depending on the degree of the desired therapeutic effects and on the patient's preoperative sensory and functional status.

■ Pain after Brachial Plexus Injuries

The incidence of pain after brachial plexus injury is reported at less than 30% for postganglionic location of the neural disruption, as opposed to 90% when the location is preganglionic.^{20,21} Pain is so intense that patients almost constantly resort to opioid consumption, frequently attempt suicide or commit self-mutilations to

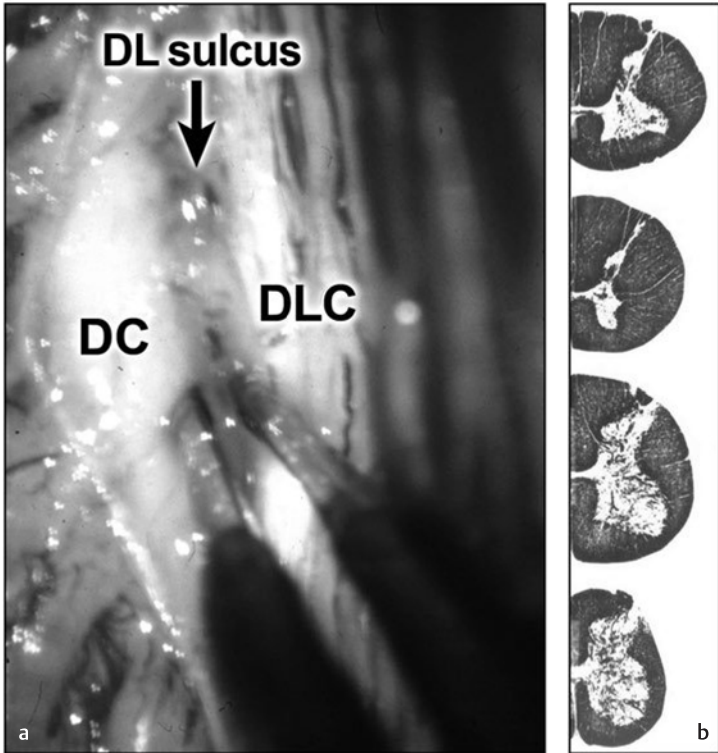


Fig. 25.2 (a) Microsurgical operative view of the dorsal horn (DH) after opening of the dorsolateral sulcus. Note the gelatinous aspect of the DH gray matter, in between the dorsal column (DC) and the dorsolateral column (DLC) white tracts. (b) Variations of shape, width, and depth of the DREZ (dorsal root entry zone) area according to spinal cord level (from top to bottom: cervical [7], thoracic [5], lumbar [4], sacral [3]). Note how at the thoracic level the Lissauer tract is narrow and the dorsal horn deep, so that DREZ lesioning, especially at that level, can be dangerous for the corticospinal tract and the dorsal column.

call caregivers' attention to their dramatic situation. From study of the effects of DREZ lesioning on an animal model of cervical plexus avulsion, we observed that after deafferentation rats performed self-directed mutilations of the autotomy type in the forelimb, and that this behavior was reversed in the group that benefited from microsurgical DREZotomy, which was not the case for those in the sham control group ($p = 0.01$).²²

A valuable preoperative appraisal of the radicular lesions can be achieved with high-resolution T2 spinal magnetic resonance imaging (MRI), electro-neuromyography (EMG), somatosensory-evoked potentials (SSEPs), and motor-evoked potentials (MEPs) investigations. Pseudomeningoceles on MRI are classically con-

Fig. 25.2 Considerations for MDT at the Cervical Level

The prone position with the head and neck flexed in the Concorde position with three-pin head holder has the advantage of avoiding brain collapse caused by cerebrospinal fluid depletion. The level of laminectomy is determined after identification of the prominent spinous process of C2 by palpation. For unilateral DREZ-operation, a hemilaminectomy with preservation of the spinous processes is sufficient to access the posterolateral aspect of the spinal cord. Laminectomy from C3–C7 included, allows exposure of the rootlets of C5–T1. After opening the dura and arachnoid, the exposed roots are dissected free by separating the tiny arachnoid filaments that bind them to each other, to the arachnoid sheath, and to the cord pia. Identification of roots can be verified by electrical stimulation at their corresponding foramen, and their functional value checked. Stimulated ventral roots have a motor threshold at least 3 times lower than the dorsal roots. Responses are in the diaphragm for C4 (the response is palpable below the lower ribs), in the shoulder abductors for C5, in the elbow flexors for C6, in the elbow and wrist extensors for C7, and in the muscles intrinsic of the hand for C8 and T1. Microsurgical lesioning is performed at the selected levels according to the preoperative program. The dorsal rootlets are displaced dorsally and medially with a hook or a microsucker to access the ventrolateral aspect of the dorsolateral. Then, an incision 2 mm in depth at 35 degree angle is made with a microknife, currently an ophthalmologic microscalpel, at the ventrolateral border of the DL sulcus. Then microcoagulations are made in a *chain*, that is, dotted manner, down into the dorsal horn. Each microcoagulation is performed—under direct magnified vision—by short-duration (a few seconds), low-intensity, bipolar electrocoagulation, with a specially designed graduated sharp bipolar forceps incremented in millimeters (graduated bipolar forceps ref: 12-30179, DREZotomy Set, Stryker Leibinger GmbH & Co.KG). The depth and extent of the lesion depend on the desired therapeutic effect and the preoperative functional status of the limb (3 mm in depth). If the laxity of the root is sufficient, the incision is performed—continuously—in the dorsolateral sulcus, thus accomplishing a sulco-myelotomy. If not, successive incisions are made ventrolaterally at entry of each of the rootlets of the root after the surgeon has isolated each one by separating the tiny arachnoid membranes that hold them together.

sidered to be indirect signs of root avulsion. In our series pseudomeningoceles were present in 31% of the patients, and almost always corresponded to total or partial avulsion of the contained roots. However, surprisingly, 50% of the partially or even totally avulsed roots were not associated with any observable pseudomeningocele; the risk of underestimating avulsed roots on imaging only should be taken into account before making a surgical decision.²³ For those patients who benefited from surgical repair after injury, the lesions described in the operative record are of major help in defining the plexus anatomical pathology.

Anatomical Findings at Surgery

Our patients referred for pain surgery all had severe radicular lesions, at least two markedly affected root levels. Altogether, 78% of the total brachial dorsal roots were impaired, 79% of which were totally avulsed and 21% either partially avulsed or atrophic. The extent of the sensory deficit corresponded to the dorsal root lesions at the intradural level in only half of the patients, indicating coexisting extrarachidian lesions, well explained by the intense stretching of the entire plexus.²³

Additional abnormalities of the spinal cord were found in 49% of our patients.²³ They consisted of marked deviation/distorsion of the cord fixed by

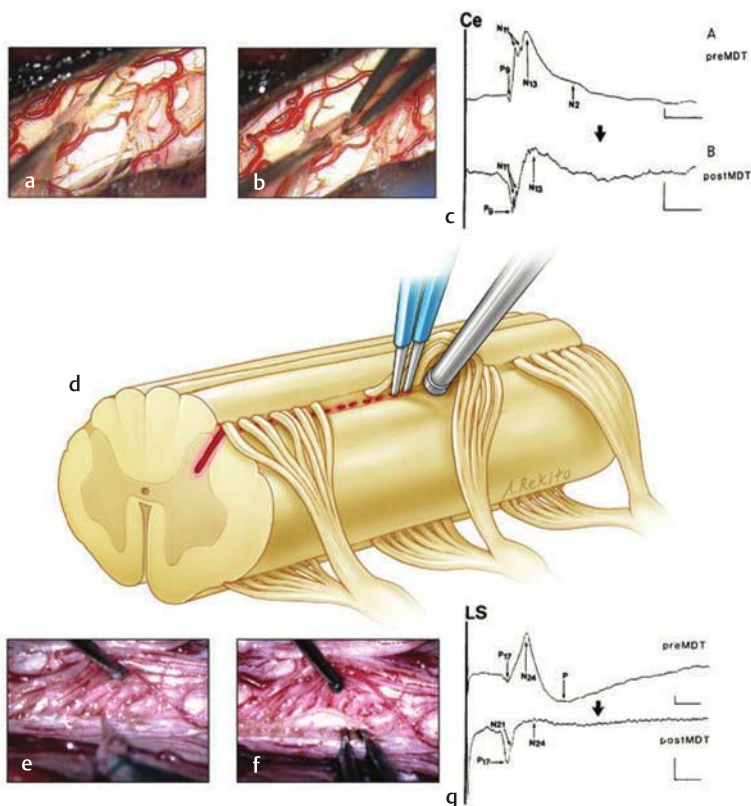


Fig. 25.3 Technical principles of microsurgical DREZotomy (MDT). (**a–c**) Operative procedure at the cervical (Ce) level, when roots are intact (**a, b**: microsurgical operative views at C7 level on right side through right hemilaminectomy). (**a**) Exposure for incision in dorsolateral (DL) sulcus. (**b**) Microcoagulations inside DL sulcus. (**d**) Drawing of MDT at cervical level. (**e–g**) Operative procedure at the lumbosacral (LS) level, when spinal cord and roots are intact (**e, f**: microsurgical operative views at L5–S1 segments on left side). Exposure of conus medullaris through a T11–L1 laminectomy and approach of the dorsolateral sulcus, on the left side in this example, by displacing the dorsal rootlets dorsally and medially. (**e**) The rootlets are held with a specially designed ball-tip microsucker used as a hook to access the ventrolateral part of the DREZ and make incision with a micro knife. (**f**) Lesioning is performed by doing microcoagulations under direct magnified vision +++. (**c, g**) Effects of MDT on the evoked electrospinalogram (EESG) recorded from the surface of the dorsal column medially to the DREZ at the C7 cervical (Ce) and the L5 lumbosacral (LS) segments, ipsilateral to the stimulation of the median and the tibial nerve, respectively, before (pre-MDT) and after (post-MDT) MDT.⁵⁷ The initial positive event P9 (for cervical), P17 (for lumbosacral) corresponds to the nearfield presynaptic successive axonal events, generated in the proximal portion of the dorsal root, the dorsal funiculus, and the large-diameter afferent collaterals to the dorsal horn. After MDT, all these presynaptic potentials remain unchanged. The larger, slow negative wave N13 (N24) corresponds to the postsynaptic activation of the dorsal horn by groups I and II peripheral afferent fibers of the median (tibial) nerves. They are diminished after MDT, in the order of two thirds. The later negative slow wave N2 (just visible in the cervical recording) corresponds to postsynaptic dorsal horn activity consecutive to the activation of group II and III afferent fibers. N2 is suppressed after MDT.

Fig. 25.3 Considerations for MDT at the Lumbosacral Level

The patient is positioned prone on thoracic and iliac supports and the head placed 20 cm lower than the level of the surgical wound to minimize CSF loss. Vertebral levels are identified with lateral X-ray from the S1 vertebra. To access the conus medullaris, a laminectomy is performed from T11 to L1 (or L2). After opening the dura and arachnoid longitudinally, the filum terminale is isolated. Identification of roots can be checked by electrical stimulation. The L1 and L2 roots are easily identified at their penetration into their respective dural sheaths. Electrical stimulation of L2 produces a response of iliopsoas and adductor muscles. Identification of L3–L5 is difficult for several reasons: (1) their exit through their respective dural sheaths is caudal to the exposure; (2) their dorsal rootlets enter the sulcus in an uninterrupted way; (3) their ventral roots are hidden in front of the dentate ligament. Stimulation of L3 produces a preferential response in the adductors and quadriceps, of L4 in the quadriceps, and of L5 in the anterior tibialis and gluteal muscles. Stimulation of the S1 dorsal root produces a motor response in the gastrocnemius-soleus group. Stimulation of the S2–S4 dorsal roots (or better, directly, the corresponding spinal cord segments at the DREZ) can be assessed by recording the bladder or more easily the anal responses by use of electromyography of the anal sphincter (or simply with a gloved finger into the rectum). Because intraoperative neurophysiologic investigations are time-consuming, we found that measurements at the conus medullaris can be sufficient in the patients who already have severe preoperative impairment of the versicoanal functions. These measurements, based on human postmortem anatomic studies, showed that the landmark between the S1 and S2 segments is situated approximately 30 mm above the exit from the conus of the tiny coccygeal root.^{3,13,14}

MDT at the lumbosacral level has the same principles as the ones at the cervical level. But at the lumbosacral level, MDT is difficult and potentially dangerous because of the rich vasculature of the conus. The dorsolateral spinal artery courses ventrally along the dorsolateral sulcus. Its diameter is 0.1 to 0.5 mm; it is fed by the posterior radicular arteries and joins caudally with the descending anterior branch of the Adamkiewicz artery through the conus medullaris anastomotic loop of Lazorthes. This artery should be preserved by being freed from the sulcus. The conus medullaris is approached through a T11–L1 laminectomy and sulcus by displacing the dorsal rootlets dorsally and medially. The rootlets are held with a specially designed ball-tip microsucker used as a hook to access the ventrolateral part of the DREZ. The main arteries running along the dorsolateral sulcus are preserved. A continuous incision is performed with a microknife. The cut is at a 45 degree angle and to a depth of 3 mm. Then, lesioning is performed by doing microcoagulations under direct magnified vision at a low intensity, in the sulcomyelotomy, down to the dorsal horn. These microcoagulations are made all along the segments of the cord selected to be operated on by means of the special sharp bipolar forceps, graduated every millimeter over 6 mm.

strong adhesive arachnoiditis, a more or less notable degree of cord atrophy on the side of the avulsion, which makes identification of the dorsolateral sulcus difficult. Therefore, freeing the spinal cord and roots prior to performing DREZ lesioning is of prime importance. Furthermore, it contributes to relief of the components of pain induced by the neck movements. Under high magnification of the surgical microscope, focal gliosis and microcysts can be found inside the dorsal horn. Such abnormalities, observed in 36.4% of our patients, were reported to be similarly important by others.¹⁰ Formation of scar and gliotic tissue at the avulsed root segments is assumed to play a role in the genesis of pain, likely by facilitating hyperactivity in the local DH neurons.

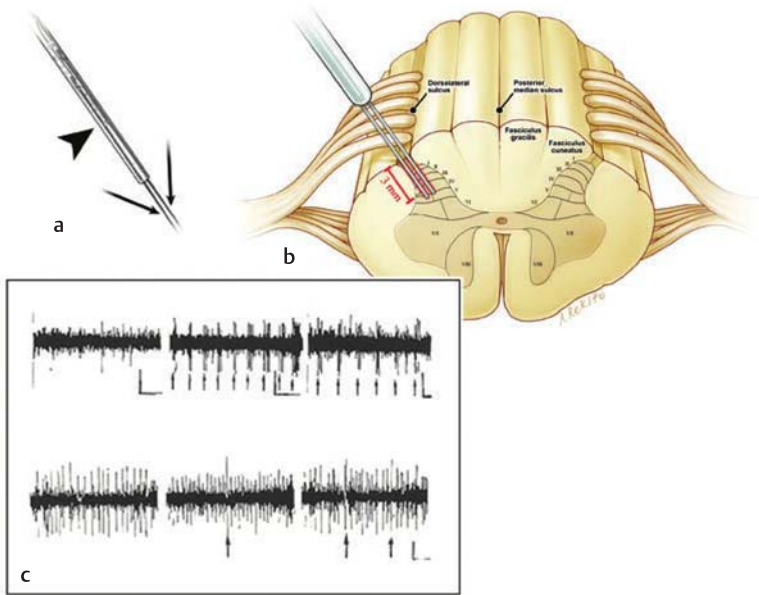


Fig. 25.4 Microelectrode recordings of deafferentation hyperactivity. **(a)** Illustration showing the floating dual, tungsten-in-glass, microelectrode of the Ainsworth-Gue-not type designed to obtain unitary recordings in the human dorsal horn. The two independent tips are separated by a distance of 300 μm , allowing for distinguishing spikes (recorded on one tip) from artefacts (recorded simultaneously on the two tips).¹⁷ The protective silicon sleeve (arrowhead) is 5 mm away from the tips, allowing them to reach the deeper part of the dorsal horn. **(b)** Schematic drawing of the microelectrode implanted into the dorsal horn following its axis. **(c)** Traces of dorsal horn microelectrode recordings in humans. *Upper trace*: normal activity. Recordings in a nondeafferented dorsal horn at the lumbosacral level (in a spastic patient). *Left*: almost no spontaneous activity (three spikes at random). *Middle*: spike discharges evoked by regular light tactile stimulation in the corresponding dermatome. *Right*: spike discharges evoked by electrical stimulation of the corresponding peripheral nerve. *Lower trace*: deafferentation hyperactivity manifested by continuous regular discharges that remain unaltered.¹⁶ Recordings in the L5 cord segment of a patient with pain due to traumatic section of the hemicauda equina from root L4–S4. *Left*: spontaneous activity of the recorded unit: continuous, regular, high frequency discharge. This activity is not influenced by tactile stimulation of the L4–S1 dermatomas (*middle arrow*), nor by electrical stimulation of the tibial nerve. The vertical bars are 50 μV ; the horizontal bars are 100 ms. These spontaneous hyperactivities, as well as the ones observed in patients after brachial plexus avulsion (*not shown*),^{16,18} are thought to be at the origin of so-called deafferentation pain, with its neurochemical substrate.⁵⁸ Such abnormalities can be reproduced in animal experiments;^{22,59,60} the clinical and electrophysiological expression of this experimental deafferentation pain can be suppressed by DREZ lesioning.^{16–18,22}

Surgical Procedure

DREZ lesioning should not be limited to the avulsed segments but extended to the remaining roots corresponding to the painful territory especially if found to be atrophic or of a grayish color (**Fig. 25.5**).²³

Under general anesthesia with tracheal intubation and short-lasting curarization, the patient is placed in the prone position, the neck flexed in the so-called Concorde position with the head maintained in a three-pin holder. This position has the advantage of avoiding brain collapse caused by cerebrospinal fluid (CSF) depletion. Through a median cutaneous aponeurotic posterior incision and unilateral paravertebral muscle division a hemilaminectomy, with preservation of the spinous processes but with ronging of their base to have enough working space, is performed ipsilaterally to the avulsion. For unilateral DREZ surgery hemilaminectomy is sufficient and lowers the risk of painful kyphotic deformity of

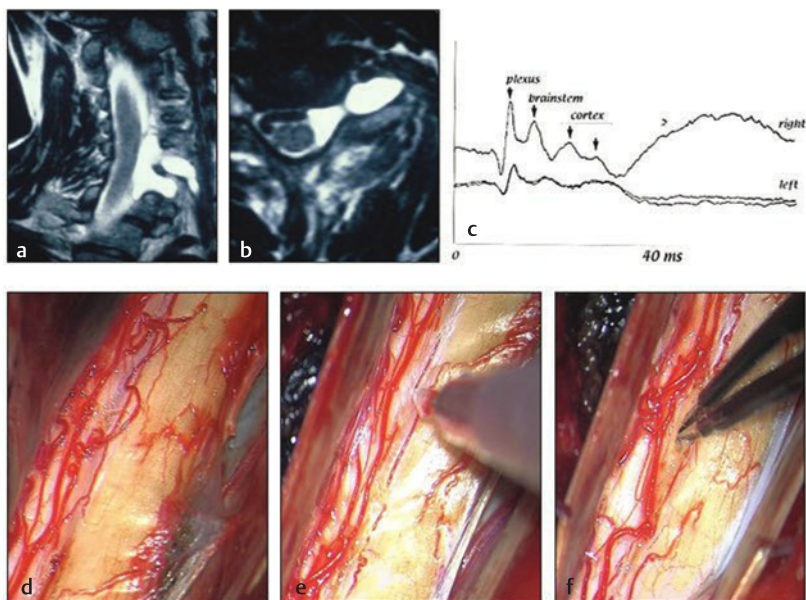


Fig. 25.5 Microsurgical DREZotomy (MDT) for brachial plexus avulsion. MDT at the cervical level for C6–T1 brachial plexus avulsion on left side. (**a, b**) T2-weighted MRI (magnetic resonance imaging) shows pseudomeningoceles at the lower cervical spine on left side. (**c**) Somatosensory-evoked potentials (SSEPs) after median nerve stimulation are abolished from cervical spinal cord up to cortex on left side, compared to right side. (**d–f**) Operative views show total avulsion from C6–T1 on the left side. (**d**) Dorsolateral sulcus (DLS) can be easily identified. (**e**) Incision into the DLS is made with a microknife. (**f**) Dotted microcoagulations into the dorsal horn, which has a gliotic aspect, are performed 3 mm inside the sulcus with the graduated bipolar microforceps.

the neck. For a total plexus avulsion, including C5–T1 roots, the hemilaminectomy is performed from C3–C7. The dura mater and arachnoid membrane are longitudinally opened. Opening is often difficult because of strong fibrotic adhesions to the cord. Pseudomeningoceles with fragile membranes are frequently found at the level of the avulsed roots.

Under the surgical microscope, the aspect (normal, grayish and atrophic, partially or totally avulsed) of all roots—both ventral and dorsal—is carefully noted. The functional status of the remaining roots can be checked by observing the muscular responses of the limb to direct electrical stimulation at 1 mA (Icare NIMBUS stimulator, Newmedic/Hemodia, Toulouse, France). Important for level identification, stimulation of the C4 ventral root causes a response in the diaphragm, palpable at the abdomen. If the dorsolateral sulcus is not clearly visible, identification should start from the intact remaining rootlets above and below the avulsed segments. The presence of tiny perforating capillaries entering the sulcus helps to determine its location. Yellow areas corresponding to old hemorrhages on the cord surface, and microcavities and gliotic tissue within the dorsal horn provide guidance for tracing the dorsolateral sulcotomy. Intraoperative monitoring of the dorsal column SSEPs and the pyramidal tract can be helpful,²³ at least until one has reached a good level on the learning curve.

As shown in **Fig. 25.5**, the first step of the procedure is dorsolateral sulcus opening. An incision is made with a microknife, of the ophthalmologic type, in the axis of the DH, 2 mm in depth and oriented 35 degrees medially and ventrally. Under magnified vision and with a sharp graduated bipolar forceps (model 12-30179, DREZotomy set, Stryker Leibinger GmbH, Freiburg, Germany, and Kalamazoo, MI, USA), dotted microcoagulations are performed every millimeter inside the DH, 3 mm in depth from the surface of the cord. Each coagulation is performed under direct vision, approximatively for 2 seconds, at low intensity of the bipolar generator. Special care is taken to locate these microcoagulations inside the limits of the dorsal horn, between the cuneate fasciculus of the dorsal column medially and the corticospinal tract laterally, to avoid impairing the sensory and motor pathways, respectively.

The same surgical principles apply for the less frequently encountered lumbar-sacral root avulsions at the conus medullaris.

Outcome

Good long-term outcome (i.e., pain relief of more than 75%) was obtained in 42.7 to 85% of the patients according to the literature review (**Table 25.1**). From our Kaplan–Meier (KM) analysis at 8 years of follow-up, a good outcome allowing withdrawal of the opioids was achieved in 85.9% of our 84 patients, of whom three fourths had a complete cure—that is, no pain and no medication.²³

Using microsurgical coagulation, RF thermal or ultrasound probes for making lesions obtained similarly good results, whereas a laser beam showed poor efficacy, likely because its lesion does not reach the deeper DH layers.

Failures and recurrences were not found to be statistically correlated with the time elapsed between injury and onset of pain or, surprisingly enough, with the duration of pain prior to surgery.^{23,24}

DREZ lesioning produced a more pronounced and complete effect on the paroxysmal than on the continuous pain component (63% vs. 26%, $p = 0.01$) in the series

Table 25.1 DREZ lesioning for brachial plexus injury: Literature reports

Reference	Technique	Number of patients	Follow-up, in years: range (mean)	Percentage of patients having >75% relief
Nashold and Ost Dahl ⁶	RF-Th	18	1–4 (1.8)	72.3
Garcia-March et al ⁶¹	RF-Th	11	1–5 (1.5)	54
Thiebault et al ⁶²	RF-Th	18	(6)	83
Campbell et al ⁶³	RF-Th	10	1–5	80
Ishijima et al ⁶⁴	RF-Th	19	(1.7)	82.4
Friedman et al ⁶⁵	RF-Th	39	1–10	67
Young ⁴⁸	RF-Th/CO ₂ laser	18/4	1–5 (4)	RF, 75; laser, 50
Kumagai et al ⁶⁶	RF-Th	7	(4.2)	42.7
Dreval ¹⁰	Ultrasound	124	(4)	87
Thomas and Kitchen ⁶⁷	RF-Th	44	1–12 (5)	79
Rath et al ⁶⁸	RF-Th	14	3–12 (6.2)	76
Samii et al ²⁴	RF-Th	47	2–18 (14)	63
Prestor ⁶⁹	Microsurgery	21	2–10 (5.6)	80.9
Sindou et al ²³	Microsurgery	55	1–27 (6)	85
Tomás and Haninec ⁷⁰	RF-Th	21	1–8	62
Chen and Tu ⁷¹	Microsurgery	40	3–10	80
Kanpolat et al ⁷²	RF-Th	14	>1	69
Aichaoui et al ²⁶	Microsurgery	29	1–10 (5)	76.9
Ali et al ²⁵	RF-Th	10	1–5 (3)	70
Taira (p.c.)	Microsurgery	53	>1	65

of Ali et al,²⁵ as well as in our study with KM analysis at 10 years of follow-up (76.2% vs. 43.1%, $p = 0.03$).²⁶ We think, nevertheless, that the presence of a continuous background of pain must not be considered a contraindication for DREZ surgery.

■ Pain after Spinal Cord/Cauda Equina Lesions

Chronic pain after spine injuries can be related not only to persisting compression or bony instability, but also to spinal cord/root nerve lesions that may generate so-called neuropathic pain, the incidence of which varies overall from 10 to 25%, according to a literature review,²⁷ and reaches 35% for the conus medullaris/cauda equina location. Classification of the pains as segmental and infralesional (that is, below the lesion) is of

practical importance. As a matter of fact, regarding segmental pain, which is the type that resides in the territories corresponding to the injury and altered neighboring segments, mechanisms may result from nerve root contusion, entrapment, or scarring, and also from the development of central dysfunction due to deafferentation or direct damage to the spinal cord neurons. These disturbances, likely due to release of the neurons from their normal inhibitory impulses or to an increase in their intrinsic excitability, lead to abnormal spontaneous patterns of discharge in the dorsal horn.²⁸ Such hyperactivities could be recorded during surgery in patients with deafferented segments of the spinal cord.^{15–18} A related hypothesis, based on the fact that cordectomy improves pain only if performed rostral to the level of the lesion,^{29,30} suggests that the origin of pain comes from the spinal cord segments just rostral to the site of injury.

When the diagnosis of neuropathic pain is retained and medical therapies have failed, DREZ surgery can be the recourse, but only if the pain is considered segmental. According to our experience, MDT has revealed effectiveness only in patients in whom pain corresponded with the level and extent of the spinal cord lesions, in contrast to the pain located in the territory below the lesion. In our series pain “below the lesion,” especially the one located in the perineosacral region, was not influenced even when DREZ lesioning was performed at the lower medullary segments.²⁷

Pain caused by lesions in the cauda equina can also be favorably influenced by MDT performed at the corresponding spinal cord segments.

Anatomic/Pathologic Data

The segmental levels to operate on are not only those injured, but also the adjacent ones if modified by consecutive pathologic processes—cavitation, gliosis, arachnoiditis, and others. The most frequent pathologic alterations due to spinal cord injuries have been well summarized by Nashold: Blunt injury to a segment of the spinal cord by a spinal dislocation results in a relatively localized spinal cord injury, whereas a gunshot wound may produce an injury that involves numerous segments above and below the injury. The initial insult is followed by central hemorrhage. After the hemorrhage resolves, small microcysts may form larger necrotic cavities which can be seen on the MRI scan. As a result of the spinal injury, not only the spinal cord is damaged, but also the adjacent tissues including the dorsal and ventral roots and the arachnoidal tissue. The arachnoidal scarring at the site of the spinal injury may be enough to tether the cord (personal communication).

Surgical Procedure

In paraplegic patients with complete motor sensory and sphincterian deficits, MDT can be done extensively on the selected segments. In patients with incomplete paraplegia, DREZ lesioning should be performed more restrictively to avoid creating additional neurological deficits. In patients with spine fractures not previously treated, surgery must start with liberation of the neural structures from the bony fragments that may occupy the intrarachidian and sometimes the intradural spaces. Frequently there may be a need for an intradural freeing from an adhesive arachnoiditis. Such a preparatory approach may be long and bloody; in that eventuality, surgery may be stopped and MDT performed in a second stage, around 2 weeks later.

Outcome

Outcome varies essentially according to the distribution of pain. As shown in **Table 25.2**, good long-term outcome (i.e., more than 75% relief) was achieved in 68 to 73% of the patients who had a predominantly segmental distribution of pain, compared with none of the patients with predominantly infralesional pain in spite of DREZ lesioning being performed down to the lower part of the cord. The clinical key for indication is the ability to differentiate the segmental from the infralesional part of the pain, which may be difficult but is of practical importance. Pain below the lesion is likely related to interruption of the ascending sensory tracts, with degeneration of fibers up to the brainstem and induction of pain generators at the supraspinal level. With regard to pain characteristics, a good outcome is better achieved on the paroxysmal and/or the allodynic components than on the permanent, often burning component. Good relief was obtained in 88% versus only 42% of our patients, respectively,^{27,31} which is similar to other studies.^{32,33} This discrepancy, which we also observed when dealing with pain after brachial plexus avulsion, is in accordance with the likely dorsal horn origin of the paroxysmal/allodynic components, and the hypothesis that the continuous component is the clinical expression of the degeneration of the spinothalamic, spinoreticular fibers.^{23,31}

Table 25.2 DREZ lesioning for spinal cord injury: Literature reports

Reference	Technique	Number of patients	Follow-up, in years: range (mean)	Percentage of patients having >75% relief
Weigand and Winkelmüller ⁷³	RF-Th	20	1–2	50
Friedman and Nashold ⁷⁴	RF-Th	56	0.5–5	Segm., 78; below lesion, 20
Young ⁴⁸	RF-Th/CO ₂ laser	20	1–5 (4)	55
Edgar et al ⁷⁵	Rh-Th	46	2–7 (3.5)	92
Sampson et al ⁷⁶	RF-Th	39	0.1–12 (3)	54
Rath et al ⁶⁸	RF-Th	22	1–13 (5)	55
Spaić et al ⁷⁷	Microsurgery	26	0.5 (0.8)	Segm., 100
Sindou et al ²⁷	Microsurgery	44	1–20 (7)	Segm., 68; below lesion, 0
Falci et al ⁷⁸	Rh-Th	41	1–6	80
Kanpolat et al ⁷²	Rh-Th	17	>1	69
Chun et al ³²	Microsurgery	38	2–6 (3.5)	Segm., 82.6
Taira ³³	Microsurgery	4	>1	57

■ Pain Resulting from Peripheral Nerve Lesions or after Herpes Zoster Infection

Pain resulting from peripheral nerve pathologies and complex regional pain syndrome (CRPS) are rare indications for lesioning surgery in the DREZ, as these pains are most often relieved by SCS.³⁴ However, in refractory situations, when the predominant component of pain is of the paroxysmal type (electric flashes) or corresponds to allodynia/hyperalgesia, MDT can be indicated because it may be effective on those components. In CRPS, the pain manifestations and the vasomotor disturbances can be favorably influenced. MDT may also be indicated for severe occipital neuralgias³⁵; the procedure can be easily performed at the C2–C3 spinal cord segments through a limited C2 hemilaminectomy. After limb amputation, two main types of pain may occur that often coexist: pain in the stump and pain in the phantom limb. DREZ surgery can be considered if SCS (and motor cortex stimulation, when phantom pain is predominant) has failed.³⁶ The existence of root avulsion should prompt recourse to DREZ surgery as a first option.

In patients who do not have important neurological deficits, lesioning must not be too extensive in depth so that the tactile and proprioceptive sensory capacities are at least partially preserved, and uncomfortable paresthesias avoided.

Postherpetic pain, well known to originate from sequelar lesions located in both the dorsal root ganglion and the corresponding dorsal horn, complicates herpes zoster infection in approximately 10% of the patients, mostly older ones. The pain syndrome is characterized by three types of algias that may be associated: permanent, burning, deep ache; electric, shootinglike paroxysms; and allodynia/hyperalgesia in the affected hypoesthetic dermatomas. There is a general agreement that DREZ surgery alleviates only the latter two components; the deep, aching pain is generally unrelieved and may even be aggravated.^{36–38}

Determination of the spinal cord segments involved may be difficult. Observation at surgery of atrophic and grayish root(s) is most helpful for their identification. When the thoracic spinal cord is the target, because at this particular level the dorsal horn is narrow and deeply situated as shown in **Fig. 25.2**, encroachment of the corticospinal tract laterally and of the dorsal column medially might occur if lesioning is not prudently performed. The literature review is summarized in **Table 25.3**.

■ Pain in Malignancies

Only a few patients with malignancies are potential candidates for DREZ surgery. Mechanisms are often mixed: somatogenic due to cancer invasion and neurogenic linked to compression/destruction or (surgical, postradiation) iatrogenic impairment of the neural structures. Criteria for selection should be very restrictive: long life expectancy, general conditions compatible with open surgery, and topographically limited pain caused by well-localized lesions. The thoracic apex syndrome is typically a good indication for MDT,³⁹ currently from C7–T2. For more extended cervicothoracic cancers, stereotactic spinothalamic tractomy,

Table 25.3 DREZ lesioning for peripheral nerve pathologies, postamputation, Hz: Literature reports

Reference	Technique	Number of patients	Follow-up, in years: range (mean)	Percentage of patients having >75% relief
<i>Peripheral nerve pathologies/complex regional pain syndrome (CRPS)</i>				
Taira (p.c.)	Microsurgery	7 (CRPS)	>1	57
Sindou	Microsurgery	42	1–15	Paroxysmal/allodynic: good outcome Continuous/deep: poor outcome
<i>Postamputation pain</i>				
Weigand and Winkelmüller ⁷³	RF-Th	7	1	14
Saris et al ⁷⁹	RF-Th	9	0.5–5	67
Kanpolat et al ⁷²	RF-Th	4	?	?
Taira (p.c.)	Microsurgery	2	>1	50
Sindou	Microsurgery	4	1–3 (2)	66
<i>Postherpetic pain</i>				
Friedman and Bullitt ³⁷	RF-Th	32	0.5–6	25
Young ⁴⁸	RF-Th	11	1–5 (4)	54
Kanpolat et al ⁷²	RF-Th	2	?	100
Taira (p.c.)	Microsurgery	2	>1	0
Sindou	Microsurgery	12	1–15	Paroxysmal/allodynic: good outcome Continuous/deep: poor outcome

open high cervical anterolateral cordotomy, or percutaneous CT (computed tomography)-guided cordotomy⁴⁰ is preferable. Further potential indications for MDT are painful conditions caused by circumscribed malignancies in the thorax, the abdomen wall, or the perineal floor, and also pain due to limited neoplastic involvement of the lumbosacral roots/plexuses. For perineal pain, midline myelotomy can be an alternative. Intrathecal morphine is the technique of choice for advanced widespread pelvic cancers. Because extensive DREZ operations at the lumbar/sacral segments would inevitably result in leg hypotonia and sphincter disturbances, for pain below the waist the procedure is indicated only if it will be limited. The relevant literature has been recently analyzed by Gadgil and Viswanathan⁴¹; data are summarized in **Table 25.4**.

Table 25.4 DREZ lesioning for pain in malignancies: Literature reports

Reference	Technique	Number of patients	Follow-up, in months: range (mean)	Percentage of patients having >75% relief
Nashold et al ^{6,80,81}	RF-Th	2 (cauda equina K)	8-4	100
Sindou and Lapras ³⁹	Microsurgery	13 (thoracic apex K)	1-30	90
Samii and Moringlane ⁸²	RF-Th	2 (breast K)	?	50
Powers et al ⁸³	Laser	3 (K)	?	100
Esposito et al ⁸⁴	Microsurgery	8 (K)	?	100
Kumagai et al ⁶⁶	RF-Th	1 (pelvic K)	2	0
Zeidman et al ⁸⁵	RF-Th	2 postradiation	29-48	100
Sindou	Microsurgery	46 (K): cervical MDT 35 (K): lumbar/ sacral MDT	1-48	87 78
Rath et al ⁶⁸	RF-Th	2 postradiation	6-8	50
Teixeira et al ⁸⁶	RF-Th	7 postradiation	2-36	85
Ruiz-Juretschke et al ⁸⁷	RF-Th	3 (cervical K)	?	33
Kanpolat et al ⁷²	RF-Th	7 (K)	?	60
Taira (p.c.)	Microsurgery	3	>1	?

■ Hyperspastic States with Pain

Because muscular tone was found very much diminished in the operated areas after MDT was performed for treatment of pain,⁴² the procedure was applied as early as 1973 for disabling harmful spasticity.^{43,44} The hypotonic effect is explained by the fact that MDT interrupts the afferents of the myotatic (monosynaptic) and of the nociceptive (polysynaptic) arch reflexes, and so deprives the somatosensory relays of the ventral horn of most of their excitatory inputs (**Fig. 25.1**).

Briefly, two groups of patients may benefit from DREZ surgery. The first group includes hemiplegic patients with severe hyperspasticity in the upper limb; MDT is performed from C5-T1 segments through a C3-C7 hemilaminectomy. The second group corresponds to paraplegic patients with disabling spasticity who are bedridden, when intrathecal baclofen is not indicated or has failed. MDT is performed bilaterally through a T11-L1 laminectomy from L2 down to S2, and additionally down to S5 when there is a hyperactive bladder with urine leakage around the catheter. For MDT in the spastic patient, intraoperative neurophysiological mapping helps in identifying root and cord levels, as well as quantifying the extent of MDT.⁴⁵⁻⁴⁷ Results have been reported in detail in the cited publications.

■ Complications

Appraisal of complications through the literature was made difficult by the lack of precise figures in a number of publications. However, it appears that complications related to the surgical approach—such as hematomas, CSF leak, infection, and meningitis—were rare and most often without sequelae. Deaths were scarce and essentially linked to precarity of the patient’s general conditions.

Neurological complications occurred as a consequence of long tract damage due to misplacement or overextension of the therapeutic lesion. There is a general agreement that the adjacent dorsal column and corticospinal tract are particularly at risk in the thoracic spinal cord due to thinness and depth of the dorsal horn at that level. At any level mistargeting may result when the dorsolateral sulcus is hard to identify because of root avulsion, myelomalacia, or severe arachnoiditis. Also, coagulation of vessels, notably at the conus medullaris, known to harbor important arteries along the dorsolateral sulcus, may be the cause of uncontrolled extension of the lesional volume. Complications compiled from the literature are summarized in **Table 25.5**.

Complications According to Level and Etiology

DREZ surgery in the cervical spinal cord, for pain after brachial plexus avulsion, for instance, entails the risk of motor weakness, ataxia, paresthesias, and other complications in the ipsilateral lower limb. In patients who retain use of the

Table 25.5 Complications reported in literature: Incidence (%) according to etiology and lesion maker

	BP avulsion 19 publ. totaling 672 pts.		SC/CE injury 7 publ. totaling 347 pts.		PN, postamputation (Hz) 3 publ. totaling 92 pts.	
RF-Rh	396 pts.	M: 2–50% S: 9–72% g-sph: 0–1.3% New pain: 0–28.5%	336 pts.	M: 3–14% S: 2–70% g-sph: ? New pain: 10%	50 pts.	M: 8–19% S: 24–50% g-sph: 1.3–8% New pain: ?
Microsurgery	148 pts.	M: 1.8–4.7% S: 1.6–14% g-sph: 0–3% New pain: 0–2.6%	112 pts.	M: 3–14% S: 2–70% g-sph: ? New pain: ?	42 pts.	M: 2.3% S: 4.7% g-sph: none New pain: ?
Ultrasound	124 pts.	M: 10% S: 15% g-sph: ? New pain: 10%	None		None	

Abbreviations: M, motor; S, sensory; g-sph, genito-sphincterian, deficits.

affected upper limb, excessive lesioning might compromise the residual function through additional sensory loss. DREZ surgery in the conus medullaris, after spinal cord injury, for instance, theoretically poses no major danger in patients who are already totally paraplegic. However, excessive lesioning could raise the height of deficits; create hypotonia, making transfers difficult; and suppress useful genito-sphincterian automatisms when these are present.

DREZ surgery may generate new pain at the borders of the operated territory. When reported, such pain was most often considered bearable compared with the original pain that led to surgical indication. Mechanisms remain putative: unmasking of previous pain, damage to the DH cells at the origin of the spinoreticular/thalamic tract, along with other possible factors.

Complications According to Lesion Maker

Although all are directed at DREZ, the various modalities for lesioning do not have the same anatomical target and the tissular lesions vary in shape and extent. Consequently, they are not all associated with the same effects and level of danger. The RF thermocoagulation procedure is performed with an electrode implanted through the pia mater; the lesion has an ovoid shape and involves the whole dorsal horn with no clear-cut limits. Lesions made by lasers, usually the carbon dioxide laser, are more superficial and have a v shape; they are often accompanied by small infarcts because of the coagulation of vessels located at the DREZ.^{48–50} The ultrasonic probe has been almost exclusively used in BPA; it has the particularity to evacuate the spongy and gliotic tissue situated in the DH apex.¹⁰ All of these methods destroy the entire DREZ and DH structures.

The MDT procedure aims at targeting predominantly the ventrolateral portion of the DREZ. It is performed under direct vision of the dorsal horn and the adjacent white matter tracts, after opening the dorsolateral sulcus—which is the key to the operation because this allows direct control of the location and extent of the lesioning process. This likely is the reason for its lower rate of side effects.

■ Conclusion

Surgery in the DREZ has its place in the armamentarium for treating pain when medications have proven insufficient and conservative neurostimulation methods have failed or are not indicated. Because DREZ lesioning is a delicate procedure and entails the risk of undesirable side effects, the criteria for indication should be very strict and based on solid comprehension of the mechanism(s) of pain in the individual patient. Provided the prerequisites are met, DREZ lesioning can be very useful for those patients who have no alternative avenue for pain relief.

Pain after root avulsion, especially at the cervical or, less commonly, the lumbar-sacral region, and segmental pain after spinal cord/cauda equina injury are prominent indications. For those conditions SCS cannot be effective because of the degeneration of the corresponding dorsal column fibers up to the brainstem,³⁴ and even more important, pain generators are located in the deafferented dorsal horn.^{15,16,18}

Pain after peripheral nerve lesions is a rare indication for DREZ surgery because SCS is generally effective. However, when SCS has failed and the main components are paroxysmal and/or allodynic, DREZ lesioning may be considered.

Pain in malignancies when limited in extent, as in the thoracic apex syndrome, may benefit from DREZ surgery, especially in patients with long life expectancy.

Pain linked to an excess of spasticity in severely disabled patients may also benefit from DREZ lesioning performed for both spasticity and pain. DREZ surgery must be considered as a second option in patients who could not benefit from botulinum toxin injections or intrathecal baclofen therapy.

Whatever the indication might be, the key point for safety of procedures in the DREZ is to perform lesioning under direct vision of the DH and adjacent tracts after microsurgically opening the dorsolateral sulcus.

DREZ surgery must be considered within the frame of the multidisciplinary armamentarium available for pain surgery.⁵¹



Video 5. Microsurgical DREZotomy for Neuropathic Pain

Editor's Comments

Dorsal root entry zone (DREZ) lesioning has proven to be one of the most important advancements in pain surgery over the past half century. There are many aspects of this procedure that are surprising.

First, this procedure seems to defy the general dictum that further injury to the central (or peripheral) nervous system is unlikely to improve pain that develops after nervous system injury. We are all well advised to not expect relief from deafferentation pain, after a destructive procedure. The outcome from DREZ lesions for nerve root avulsion pain does not seem to follow this rule. Perhaps the only other exception to this guideline is trigeminal rhizolysis (radiofrequency, radiosurgical, glycerol, or surgical) for trigeminal neuralgia. In fact, the latter example may not be a violation of the rule, since the efficacy of these denervating procedures may depend simply on diminishing, or eliminating, triggering stimuli rather than changing the generator of the pain. Thus, DREZ lesions may represent a unique category of surgical procedures for deafferentation pain.

Second, the generator of pain from nerve root avulsion must, to a large extent, exist within the spinal cord dorsal horn. This seems contrary to the general notion that once a nervous system injury occurs, whether peripheral or central, the sensory system central to the injury is physiologically altered, to some extent irreversibly, and that there is no unique "focus" of pain generation. This is probably true for spinal cord injury pain that is perceived below the injured spinal segment, as Dr. Sindou points out in this chapter. Something about plexus avulsion pain is different from spinal cord injury pain, in that despite complete deafferentation distal to the injury, pain relief can be achieved by DREZ lesions in the former, but not the latter.

Finally, the success rates for DREZ lesions in the problem of plexus avulsion pain, the most common indication for the procedure, are surprising. Complete cure (no pain, no medication) of these pains, which are otherwise almost completely refractory to pharmacological or surgical intervention, in three fourths of the patients is, to say the least, quite remarkable.

I do not have the experience of Professor Sindou in the treatment of pain of malignant origin, or spasticity, using DREZ lesions. What experience I do have in treating brachial plexus and lumbosacral plexus avulsion pain, and spinal cord injury pain, is completely congruent with Dr. Sindou's narrative. He invented this procedure, has provided consistently forthright assessments of its outcomes, and continues to teach us how it might be best employed. The world of pain surgery is deeply in his debt.

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